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HISTORY OF THE CLASSIFICATION OF GASTRO-INTESTINAL DISEASES IN ANCIENT AND MODERN TIMES *

ISAAC A. ABT, M.D.
CHICAGO

The nutrition of the new-born infant is not a new subject, and the diseases of the digestive tract and the intestinal disturbances of infancy, we may assume, are as old as the race.

It has been thought that on this occasion an historical retrospect of gastro-intestinal disorders would be of interest; especially at this time when new hypotheses are appearing in certain quarters as to the nature of gastro-intestinal disorders in infants, and since some of the views which were held a decade ago are being subjected to sharp attack. It is obvious that a scientific classification depends on exact knowledge.

HISTORICAL

In reviewing the literature from the earliest period to the present time, we find great variation in clinical description and in the interpretation of the gastro-intestinal disturbances of infancy. The classifications, in so far as they have been made, have been influenced by the prevailing thought of the period. In ancient and medieval times when medical knowledge was simple and when fundamental facts in anatomy, pathology and physiology were meager or unknown, classification of disease was elementary and imperfect. In the anatomico-pathologic period the profession was possessed by the thought that every disease must have not only a distinct anatomic location, but must be represented by gross anatomic and histologic changes in tissue and cells. Virchow pronounced the dictum that every physiologic type must have its pathologic prototype. With him, cellular pathology was sufficient to explain disease processes of almost every kind. With the advent of the bacteriologic era, the flora of the gastro-intestinal tract was most assiduously studied. Normal and abnormal organisms were discovered, but it was soon found that bacteriologic investigation had its limitations. It was settled for all time that some gastro-intestinal diseases were definitely due to bacteria, but it was shown also that there was a group of infantile intestinal

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diseases which seemed to bear no definite relation to the nature, number or kind of bacteria. Then began the investigations of food and the physiology of their digestion, perversion of digestion, studies in metabolism, and the effect of anomalies in the constitution of the individual in so far as they could affect digestion and nutrition. Classification, then, of the gastro-intestinal diseases of infancy at any period, can consist only of a systematic arrangement of the knowledge of that period. A real classification can be made only when a sufficient number of facts are accumulated to explain the nature of the disease processes. It is evident, too, that the various arrangements into groups of diseases reflect the prevailing medical opinion of the time. Progress has been slow; the labor and energy which has been expended in the study of this disease group has not been in vain and need not be done over again. If the anatomico-pathologic findings are insufficient to explain the facts, then possibly the bacteriologic investigations may shed more light. If the latter are insufficient, then possibly chemical and physiologic investigations may assist in unraveling the mysteries of the difficult problems which present themselves. It is important, then, that we study this disease group in historical retrospect, lest we be carried away too far by the enthusiasm of the present, forgetting the labors which have already been performed, the facts which have been accumulated and the positive and negative evidence which has been presented to us by those who have preceded.

Diseases of the mouth, especially aphthous stomatitis, were described by Hippocrates, and in more recent times by Billard. Thrush was recognized by Soranos about 200 A. D. Nutrition of infants and children was discussed by the ancient East Indians. They directed that emetics be administered once weekly and laxatives once monthly. Historians tell us that East Indian physicians found that breast milk was not secreted for the first three days. During this interval they advised that the infant be given salt and butter, or honey — with the view of producing mild catharsis. In case of failing mother's milk, a wet-nurse was to be secured when the infant was 10 days old. They cautioned, however, that the wet-nurse should undergo a careful examination by a physician. It appears that this necessary evil, wet-nursing, was in vogue among the well-to-do class in ancient India as it is with us to-day.

After the first month it was the custom to give the infant a sweet paste to suck. The infant was weaned at the sixth month, we are told, and after this time was fed rice, sour milk, and after the third year meat from goats and fowl, with butter and honey.

Very important are the writings of Soranos (Ephesian) 200 A. D. Chapters 24 to 40 of his works reflect the knowledge of children's diseases of his time, but also contain improvements and suggestions of his own. He advises, for example, that the mother should not nurse too frequently

or too long, and during the night the mother is directed to nurse the infant as little as possible. Large wet-nurses produce better milk than small ones. He describes finger-nail and water tests for breast milk. Onions, fatty and highly spiced foods are condemned because they will produce deleterious effects in the breast milk. Excessively fat milk, Soranos thought, was capable of working injury to the infant because it could not find its way through the small intestinal apertures. Almost modern seems his caution not to rock or shake the baby after nursing because it produces a condition like sea-sickness. He warns against frightening children, and devotes two chapters (39 and 40) to dentition and weaning. It is probable that the disorders of dentition were the subject of discussion in his day. He held, however, that scarification of the gums was unjustifiable. He believed in simplified therapeutics and discarded the then prevalent custom of administering urine as a remedy, and warned against too vigorous manipulation in mouth washing. In the treatment of thrush the bare finger should not be used, but should be well covered.

Dr. Ludwig Unger in 1904 compiled a monograph entitled "The Child Book," Bartholamäus Metlinger (1457-76), being a history of children's diseases in the middle ages. This is the earliest German pediatric publication. The work is of doubtful scientific value, and reflects the scholasticism and the mysticism of the middle ages. The second part of Metlinger's work treats of the nourishment of children, and also of the infant who cannot nurse at the breast; it tells how a wet-nurse may be secured and how the infant may be weaned. He believes that the milk is produced from the blood of the mother which flows through the veins into the breast and milk is formed in some indefinite way. Concerning the digestive disturbances of infants he says that indigestion is produced among children by an excessive quantity of milk which they cannot take care of.

Somewhat later, 1540, Sebastiano Austrio considered under separate headings flatulence, humid or white flow, diarrhea, cholera and diarrhea and vomiting, and Thomas Phayre in 1551 in a book, "The Regiment of Lyfe; Whereunto is Added a Treatise of the Pestilence with the Booke of Children; Newly Corrected and Enlarged"; "Febleness of the Stomacke and Vomitings" gives him opportunity for some philosophic thought. "Colyke and rumblings of the guttes cause the child restlessness. He crieth and frettith itself and many times also makes urine by reason of winde that oppresseth the neck of the bladder."

The period that we are referring to seems to be marked by the lack of uniformity in classification. Various writers show no common view concerning the underlying causes of diseased conditions. A vague symptomatology for the most part was the basis for description. Thus Sylvius

Francisco mentions "gripes of the belly due either to wind or from sour and sharp humours."

Gaulteri Harris, 1671, thinks that all the symptoms of gastro-intestinal disorders owe their origin to acid products in the body. He gives no classification of the gastro-intestinal diseases, though speaks of vomiting and diarrhea in a general way, caused by the presence of acids. Even in modern times the theory of acid intoxication has played an important part in explaining the pathogenesis of gastro-intestinal disorders.

A little later we find John Astruc, 1746, attempting a classification on the nature or appearance of the stools. Thus he speaks of stercoral, cœliac, lienteric and dysenteric stools. Worms receive a great deal of attention in this treatise, and he finally concludes that worms are not due to spontaneous generation, but that they are produced from eggs.

Nicholis Rosen von Rosenstein, a Swedish physician (1706 to 1773), a professor in Stockholm, was an active and versatile writer on practical subjects. He followed in the footsetps of Lars Roberg. Rosen gave instruction in clinical medicine and his "Diseases of Children" was popular in his own country and in England for a long time. Rosen divided the gastro-intestinal diseases into constipation, rectal prolapse, flatulence, abdominal pain and colic. The classification is based entirely on symptomatology and he proceeds to subdivide diarrhea into fourteen different varieties.

During the eighteenth century the knowledge concerning pathology and nature of gastro-intestinal diseases of children remains vague. The remarks of Roswell Park (An Epitome in the History of Medicine) are pertinent. He says that in reviewing the theories, as well as the lives of the medical luminaries of the eighteenth century, one experiences a feeling of mingled respect and disappointment; respect for the devoted way in which they worked, and sought for the truth, and disappointment at so much waste of intellectual power and labor. The lesson is taught also, and should be impressed, that in all the so-called new systems, old principles for the most part reappear, and that the labors of the past are rarely so deliberately consulted as to guard against repetition and revamping of the theories that had long before been proved futile.

William Moss, 1781, wrote an essay on "The Management and Nursing of Children in the Earlier Period of Infancy," and lays great stress on "Gripes, with or without Looseness."

Eberle, 1783, was the first to consider in detail the colic of infants.

Michael Underwood, 1789, classified the gastro-intestinal diseases in a purely symptomatic manner. He says that "the true watery gripes is esteemed the most dangerous of all purgings and is usually thought fatal; if the purging continue for a few days the stools are very thin and numerous. The child looks wretchedly and everything it takes runs

almost immediately through it." This condition is not unlike the cholera infantum of the later writers.

Christoph. Girtanner, 1794, refers to the work of Benjamin Rush on infantile cholera.

In 1773 Dr. Benjamin Rush gave a description of the symptoms of cholera infantum and the discussion of its etiology and treatment. He thought that the disease seldom appeared in Philadelphia until the middle of June, generally continued until near the middle of September; and its frequency and dangers are always in proportion to the heat of the summer. The observations of Rush were soon corroborated by his contemporaries and his successors in America and abroad.

THE ERA OF SCIENTIFIC METHODS

The nineteenth century is distinguished by the introduction of more purely scientific methods. This is the century of Darwinism, Spencer, Huxley and Heckel. Roswell Park says that the discoveries of botany, the results of better knowledge of natural history and more accurate habits of study have influenced modern progress not a little, and have led to better classification and broader knowledge. The development of the laws of physics, more accurate knowledge of physiology and anatomy, extension of the knowledge of chemistry and zoology, have exerted a wide influence on the methods of study and on the introduction of modern scientific thought.

John Cheyne, 1802, wrote a very painstaking account of icterus and described a condition which he designates as green scours, and atrophia ablactatorium, or weaning brash. The latter was an atrophic condition caused by the weaning of children too suddenly and at an unfavorable season of the year. He performed autopsies on these cases and records that they correspond to cases of cholera infantum described by Rush.

In 1821 Adolph Henke published infant mortality statistics. He believed that half of all the children born died before the age of 10 years.

C. M. Billard, 1828, was trained in the anatomic school of France, and made a specialty of the diseases of children, and his publications were important and epoch-making. He divided diseases into a group which considered the functions of the organs, and into a second group which treated of disorders associated with organic lesions. He described diarrheas and the gastric and intestinal disorders.

William Potts Dewees, 1836, an American writer, divided the diarrhea of infants into seven varieties and described each in considerable detail. To illustrate: In the biliary form of diarrhea the stools are loose, copious and of a bright yellow or green. The mucous variety he thinks is caused by a sudden checking of perspiration, or sudden application of cold to the body or feet. He closes with a consideration of cholera infantum and asserts that the disease is peculiar to this country.

Alois Bednar published an important work in 1850. He discusses diseases which are based on abnormal processes, or decomposition of foods ingested into the alimentary tract, and disorders of the gastro-intestinal tube which are based on anatomic changes of the tissues. Bednar's classification was based on wide clinical observations and pathologic investigations. His descriptions were comprehensive and exerted a strong influence on the thought of the period.

The book of Barthez and Rilliet, 1855, indulged in considerable speculation as to the nature of softening of the stomach; and considers softening of the stomach and intestines separately. They divide the diseases into three sections. The first, primary catarrhal; second, secondary catarrhal, and a third group in which the causes are of doubtful catarrhal origin but are non-inflammatory.

J. Forsyth Meigs, 1848, recognized the fact that the diseases which involve the stomach are usually associated with similar lesions of the intestinal tract, and are little likely to occur alone. He makes two classifications, one functional and the other degenerative; the latter attended with appreciable anatomic lesion.

J. Lewis Smith, 1869, considers non-inflammatory diarrhea, and the intestinal inflammation of infants.

Edward Henoch in 1862 discusses dyspepsia and divides it into acute and chronic forms. Of the intestinal diseases he considers cholera infantum, catarrhal diarrhea, dysentery and constipation.

The climax of this period is represented in greatest detail by the writings of H. Wiederhofer, 1880. Wiederhofer was associated in his work with Professor Kundrat of the Pathological Institute of Vienna. Wiederhofer was inspired by the clinical insight of his Vienna predecessors; Kundrat by the excellent traditions of Rokitansky, and of the Pathological Institute of Vienna. These writings represent the anatomico-pathologic era in the classification of gastro-intestinal diseases of infants. The classification is a long and painstaking one, including diseases of the stomach and intestines. Wiederhofer in an ingenious way endeavors to reconcile pathology and anatomy with general symptomatology and his own bedside observations. The observations were acute and unerring; clinical descriptions were painstaking; there were wide gaps in the knowledge concerning causation and interpretation.

Jacobi in 1887 contributed a valuable monograph classifying diseases into gastric and intestinal affections, and in a paper published somewhat later considers the infective causes of intestinal diseases, particularly dysentery. In his earlier publication he advocates the use of cane sugar in lieu of milk sugar. He suggests, under certain conditions, the use of beef or mutton broth, and takes occasion to say that it is not the chemical

formula alone which determines the rank of a substance as a nutrient. The digestibility of the food must be considered.

Biedert, 1878-1894, gives it as his opinion that the cause of gastro-intestinal disease of infants in the majority of cases is due to food. He lays great stress on a condition which he terms fat diarrhea, and he recommends a classification based on the appearance of the stool. He considers in detail overfeeding, the diseases due to decomposition of proteins, diseases due to excessive fat feeding, and a group of disturbances produced by excessive sugar, especially as it occurs in breast milk.

Baginsky represents the transitional period from the anatomic-pathologic to the bacteriologic era. He thought that micro-organisms were not specific; nevertheless, the common saprophytic bacteria of the intestines were capable of taking on virulent activity and becoming the pathogenic microorganism; though foreign bacteria may gain access to the gastro-intestinal tract these bacteria may, according to Baginsky, wander from the intestine into other organs, such as the kidney; they are rarely found in the circulating blood. He says the severest disturbances are caused by the action of bacteria on the foodstuff, producing fermentation and toxic products.

The labors of Booker, of Lesage and Escherich did not show any specific microorganisms for the diarrheal diseases. Escherich considered that gastro-intestinal diseases might be due to ectogenic and endogenic causes. The ectogenic gave rise to toxic gastric catarrh, while the endogenic caused infection of the chyme, with dyspepsia. After all the labor no specific organism was found.

Bacteriologic research was of value, though it was negative. The bacteriologists themselves conceded that other lines of research might yield greater reward. Investigators turned their attention from bacteriology to the chemistry of food, and studies in the physiology of digestion. Experiments in metabolism were inaugurated in many quarters.

MODERN CLASSIFICATIONS

The American Pediatric Society in 1894 offered a classification. The classification is very extensive. The enteric diseases are divided into non-inflammatory and inflammatory. The non-inflammatory may be mechanical, such as dilatation of the colon, prolapse of the bowel and polypi. The classification is very extensive, though in many instances and of necessity non-commital. It is complicated and seems difficult of practical application at the bedside.

From all that has gone before, it is evident that the digestive disturbances of nurslings were in the past classified variously according to the progress of physiology, pathology and bacteriology. The oldest classification was based on symptomatology. One would suppose that pathologic research and knowledge would have led to a simpler classification. This,

however, was not the case. Referring to the classification of Wiederhofer in 1880 and Baginsky in 1884, it is seen at a glance that they were not inclusive. The pathologic knowledge was vague and in most cases indefinite. It was also observed that one and the same pathologic lesion was associated with a varied or inconstant clinical course. Thus it was noted that inflammatory conditions as a basis for classification of the digestive disorders in infants resulted in disappointment. Terms like catarrh, gastric catarrh, intestinal catarrh, gastritis, enteritis, are used in a confusing way and require considerable elucidation in every instance, and pathologic conditions were persistently assumed which did not really exist. As improvement in pathologic investigations and technic occurred, lesions which were supposed to exist could not be confirmed. Although bacteriologic research furnished interesting and valuable information concerning normal process in the digestive tract, it did not afford data for classification of pathologic process of gastro-intestinal disorders. Thus bacteriologic classification failed. Some authors attempted to refer all digestive troubles to bacterial action, and when the ectogenic causes of diseases could be excluded, then the normal endogenic microorganisms of the intestinal tract were supposed to be capable of taking on pathologic activity and producing the disease in question. Czerny and Keller insist that aside from the intestinal disorders which are explained by pathology or by bacteriology, there are many disturbances of nutrition which could be understood only by a knowledge of infantile metabolism. All three modes of investigation are of the greatest importance and none should be used exclusively while others are neglected. Since the most enlightened students of this disease group cannot accept a bacterial cause for the entire group of gastro-intestinal diseases, it is necessary to divide them into two classes:

1. Those diseases which are evidently bacterial in origin.
2. Those in which there is no evidence of a bacterial infection.

It is a well-known fact that bacteria take part in all the normal processes of digestion. This does not imply that they are always causal factors in disease. This very fact led Czerny and Moser in 1894 to make a general classification into two groups — dyspepsia and gastro-enteritis. Czerny and Keller in their Ernährungsstörungen say that the names of the two groups were unfortunate because they have been employed by other authors in former times with various meanings. Nevertheless, the basic idea was accepted and was finally made use of in the classification of the Pediatric Section of the Paris International Medical Congress, 1900. Czerny and Keller insist that the digestive disorders of infants should be separated from those of older children. This was in contrast to the older writings of Wiederhofer and Baginsky. Czerny and Keller propose the following classification:

1. Digestive disturbances *et alimentatione.*
2. Digestive disturbances *ex infectione.*
3. Digestive disturbances through congenital anomalies.

CLASSIFICATION OF FINKELSTEIN

The historical résumé would be altogether incomplete without a brief reference to the recent teachings of Finkelstein; therefore, without going into detail into material which is fresh in every one's mind, I shall in the briefest space possible give a short synopsis of his contribution. His classification is the last one to be offered, it is based on researches into the chemistry of food, studies in metabolism, together with careful clinical analysis. He divides the subject as follows:

1. Weight disturbances characterized by a diminished tolerance for milk, especially milk fat. The infant does not thrive. The weight is stationary as a rule. The bowel movements are hard, dry and formed.

2. Dyspepsia is a more severe disease manifestation than the foregoing. It is characterized by a diminished tolerance not only for fat but also for carbohydrates. Such children do not gain in weight; they tend to suffer from diarrhea, and fail to increase in weight even if the diet is increased, constituting the so-called paradox reaction.

3. Decomposition, a condition characterized by marked loss in weight, low temperature, slow pulse. The loss in weight continues even when considerable food is administered; easily infected, or infection takes place readily. A marked intolerance for fat and carbohydrates is present. These infants are subject to secondary infectious processes, as furuncles, abscesses, etc.

4. The next clinical group is alimentary intoxication, characterized by fever, disturbances of consciousness, albuminuria, rapid superficial breathing, great intolerance for food, particularly for sugar and other whey constituents.

In considering the disturbances produced by breast milk, he refers to underfeeding as well as the dyspepsia produced by overfeeding. Disturbances of nutrition on account of congenital perversions of the constitution receive attention; for instance, he discusses the neuropathic constitution, the exudative diathesis and errors in development as factors influencing digestion and nutrition. He also considers the effect of quantitative changes in food which refers to overfeeding and underfeeding. Passing next to qualitative changes he pays particular attention to the injuries produced by starch. He devotes a chapter to parenteral infection and insists that infants suffer infections because food tolerance has been lowered by disturbances of nutrition, the lowered resistance which results in consequence making infection possible. He concludes with a chapter on the infections of the gastro-intestinal tract though he believes that parenteral infections are more frequent than genuine gastro-

intestinal infection. He concludes by saying that knowledge of the gastro-enteritis of infants, so far as bacteriologic facts are concerned, is only slightly developed, and consequently present views are subject to considerable changes in the future. He devotes a chapter to infections, gastro-enteritis, typhoid, septic infection and concludes with a short chapter on colitis.

DEDUCTIONS

One cannot fail to be impressed with several salient points as he skims hastily over the progress of the ages: First, the changing views depend on advance in knowledge and the variation in human thought during different periods. The sound logical reason of one age has yielded to mysticism, speculative philosophy and humoral pathology of another age. In some instances those whom we consider the ancient saw more clearly than medieval thinkers. Whither are we drifting now? Are we standing on firm foundation, or are we again on the quicksands? Has all the work in cellular pathology been completed? Has it shed all its light? Is the biologic study of the gastro-intestinal tract forever a closed book? Are all of the questions that confront us in the solution of this group of disorders to be disclosed by chemical study of food and the more complicated inquiries into the metabolic changes? The danger is lest we drift too far. Is it not well that occasionally we take inventory of the debts which we owe the past as well as the contribution of the present? We see light from the researches of to-day, though the illumination is not sufficient to permit us to say that all darkness has vanished.

104 South Michigan Avenue.

STUDIES IN THE BACTERIOLOGY OF THE ACUTE INTESTINAL DISEASES OF INFANCY*

BORDEN S. VEEDER, M.D.
ST. LOUIS
AND
ROBERT KILDUFFE AND O. T. DENNY,
PHILADELPHIA

The relationship of bacteria to the acute intestinal diseases of infancy is a study that has attracted many workers and yet to-day the problem is far from being answered in a satisfactory manner. Several reasons exist for the indefiniteness of the views that are held at the present time, chief among them being the variance in the bacteriologic findings, the failure to distinguish between the presence of bacteria and the action of bacteria, and the unsuccessful attempts which have been made to correlate the laboratory findings with the different clinical types of intestinal disturbance. Much of the early work is at present of value only for its historical interest, as the more recent advances in bacteriology have shown the earlier differentiation of organisms and methods of study to have been incomplete.

In the earlier days of bacteriology numerous organisms were reported as the cause of dysentery epidemics. In 1888 Chantemesse and Widal[1] described a short bacillus which was later claimed by Widal to be identical with *B. dysenteriæ*. Maggiori[2] reported several organisms from Italy in 1892, and in the same year Ogata[3] described a Gram-positive organism in Japan. Two years later Silvestri[4] contributed a diplococcus and Arnaud[5] a colon bacillus-like form. In 1895 Celli and Fiocca[6] reported the streptococcus and an organism resembling the colon bacillus which Celli[7] subsequently claimed to be identical with the Flexner type of dysentery organism. Janowski[8] reached the conclusion from his studies that no one organism was the cause of dysentery. This same view was held by those workers who studied the intestinal flora of infants.

*From the Department of Medical Research, University of Pennsylvania, Philadelphia.

1. Chantemesse and Widal: Gaz. méd. de Paris, 1888, No. 16.
2. Maggiori: Centralbl. f. Bakteriol., 1892, xi, 173.
3. Ogata: Centralbl. f. Bakteriol., 1892, xi, 264.
4. Silvestri: Riforma med., 1894, No. 22.
5. Arnaud: Ann. de l'Inst. Pasteur, 1894, viii, 495.
6. Celli and Fiocca: Centralbl. f. Bakteriol., 1895, xvii, 309.
7. Celli and Valenta: Centralbl. f. Bakteriol., 1899, xxv, 481.
8. Janowski: Centralbl. f. Bakteriol., 1897, xxi, 88.

Among them Booker,[9] who studied the subject for a number of years, stands out most prominently. From a bacteriologic standpoint he separated out two groups of cases, one a "bacillary type" which he considered as probably due to the *Proteus vulgaris*, and the other a group of more severe cases in which streptococci were found. Escherich[10] and his pupils likewise made quite extensive studies but found no one organism in constant association. By some of the earlier writers the *B. coli* was considered of etiologic importance and even more recently Apfelstedt[11] has written in support of this view. Williams and Cameron,[12] Escherich,[10] Cook,[13] Cooley[14] and others have described the *B. pyocyaneus* in association with epidemics of dysentery in infants, and other organisms such as the *B. acidophilus* (Salge[15]) and *B. perfringens* (Sittler[16]) have been reported. Kendall and Smith[17] in 1911 reported three cases in which they found *B. aerogenes capsulatus*, the association of which with the dysenteric condition they consider of importance. Practically all of these studies were made before the discovery of the dysentery bacillus by Shiga in Japan and Flexner in Manila, and hence no conclusions of any importance can be drawn from them.

DYSENTERY ORGANISMS

The discovery of the *B. dysenteriæ* in epidemics of dysentery in various parts of the world, and the subsequent attempts to correlate these organisms with the various types of intestinal disturbance peculiar to infancy, necessitates a special study of this group of organisms. It is unnecessary for our purpose to repeat the history and early controversies in regard to the identity and unity of the organisms, as it is at the present time accepted that there is a group of closely related bacteria differing slightly by cultural and agglutination reactions which may be grouped together under the term *B. dysenteriæ*.

The following classification has been quite generally accepted, and although a number of other types and subvarieties have been described, it suffices for our purposes. It may be that some of the subvarities are mere mutations as Müller[18] claims to have observed.

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9. Booker: Arch. Pediat., 1890, vii, 92; Johns Hopkins Hosp. Rep., 1897, vi, 159.
10. Escherich: Wien med. Wchnschr., 1897, x, 917; Deutsch. med. Wchnschr., 1898, xxiv, 633; Jahrb. f. Kinderh., 1899, xlix, 137.
11. Apfelstedt: Therap. Monatsh., 1906, xx, 473.
12. Williams and Cameron: Jour. Path. and Bacteriol., 1896, iii, 344.
13. Cook: Jour. Am. Med. Assn., 1904, xliii, 1933.
14. Cooley: Jour. Am. Med. Assn., 1908, l, 607.
15. Salge: Der akute Dünndarmkatarrh des Säuglings, Leipsic, 1906.
16. Sittler: Centralbl. f. Bakteriol., 1908, Orig. xlvii, 551.
17. Kendall and Smith: Boston Med. and Surg. Jour., 1911, clxiv, 306.
18. Müller: Centralbl. f. Bakteriol., 1904, Ref. xlii, Part 57.

FERMENTS

		Mannite	Dextrose	Saccharose	Maltose	
A	I	—	+	—	—	Shiga or Shiga-Kruse "New Haven."
	II	+	+	—	—	'Y' (Hiss and Russell) "Seal Harbor."
B	III	+	+	+	—	Strong (Manila).
	IV	+	+	+	+	Flexner or Flexner-Harris "Baltimore" (Duval).

The first variety — Shiga — is culturally distinguished from the remaining three (Group B) by the fact that it does not ferment mannite with the production of acid, and hence is sometimes called the "non-acid" or "alkaline" variety. Type I is claimed by Kruse[19] to be the etiologic organism in true epidemic dysentery and he speaks of the organisms of Group B as "pseudo-dysenteric organisms." As the organisms of Group B have been isolated from cases of severe epidemic dysentery, this term has found but little acceptance. But it is quite generally conceded that, as a rule, the cases from which the Shiga organism has been isolated are clinically more severe and for this reason the term "para-dysentery" has been suggested and applied to Group B by Park,[20] Castellini[21] and others. The toxins of the Shiga variety are partly soluble or exogenous, and those of the acid varieties are fixed in the organism or endogenous. As the toxins of the Shiga variety are far more toxic than those of the acid variety, the names *"giftigen"* and *"giftarmen,"* respectively, are used by Lentz[22] to distinguish the groups. According to Doerr no constant or characteristic toxin formed by the Flexner organism (Group B) has been found as far as animal experimentation has shown. A large part of the work on serum reactions — and this applies particularly to the studies on infants — is worthless because of the failure of early workers to distinguish between varieties. Each of the four types forms an homologous or chief agglutinin and at the same time heterologous or partial agglutinins for the other types. The serum distinctions are more marked between the acid and alkaline varieties than between the different members of Group B. According to Lentz[22] the Flexner or "Y" variety may be agglutinated by the serum of a normal person in dilutions of from one-thirtieth to one-fiftieth, and hence partial agglutinins may be quite high. In artificial immunization partial agglutinins are formed for heterologous varieties which interfere considerably and which differ with the animals used. Thus the horse is not suitable for immunization with the varieties of Group B. The early technic for isolation of the organisms was laborious and possessed many difficulties and is one factor

19. Kruse: Deutsch. med. Wchnschr., 1907, xxxiii, 292.

20. Park: Jour. Infect. Dis., 1905, Suppl., 295; Park, Collins and Goodwin: Jour. Med. Research, 1904, xi, 551.

21. Castellini: Jour. Hyg., 1904, iv, 495.

22. Lentz: In Kölle u. Wassermann's Hand.d.path. Microorg., 1909, Part 2, 391.

which has led to such marked differences in results. More recently the use of Endo's[23] medium which has been modified by Kendall and Walker[24] has come into use and this special medium has simplified the technic considerably.

In the summer of 1902 Duval and Bassett[25] undertook the study of infantile diarrheas at the Thomas Wilson Sanitarium in Baltimore with the purpose of determining the presence or absence of the dysentery organism. Positive results were reported in forty-two out of fifty-three cases, or approximately 80 per cent. Wide-spread interest followed this report as it was inclined to interpret their work as showing that "summer diarrhea" was an infectious process due to the *B. dysenteriæ*. The following summer an extensive investigation was undertaken in Boston, New York, Philadelphia and Baltimore under the direction of the Rockefeller Institute.[25] In all 412 cases were studied, and of these 279, or 63.2 per cent., were reported as showing the presence of some type of the *B. dysenteriæ*. The following table, which gives the results of the individual workers, shows that quite marked differences exist in their findings.

	No. of Cases	B. Dys. No.	Present Per cent.
Duval and Schorer	79	75	94
Bassett	73	51	70
Wollstein and Dewey	62	48	78
Cordes	51	26	51
Waite	47	19	40
Kendall	31	29	93.5
Lewis	21	11	52.5
Gay and Stanton Hospital	20	13	65
Gay and Stanton Tenements	28	7	25
	412	279	63.2

Examining more closely the tables of Duval and Schorer, who with Kendall report results which would lead one to believe that the organism was almost constantly present and the only organism of specific importance, we find that in twelve of the positive cases but one colony was obtained from an average of about sixteen plates. In only some twenty of their cases were a number of colonies isolated.

In addition to the Rockefeller investigations a number of independent studies have been reported. In the winter of 1902-1903 following Duval's and Bassett's first report, Wollstein[26] examined the stools from 114 cases of dysentery from the wards of the Babies' Hospital and Foundling Asylum in New York, and found the organism in thirty-nine cases, or 34 per cent. Wollstein does not give any idea as to the numerical presence of the organism.

23. Endo: Centralbl. f. Bakteriol., 1904, xxxv, Orig., 109.
24. Kendall and Walker: Jour. Med. Research, 1910, xxiii, 481.
25. Studies from the Rockefeller Institute, 1904, ii.
26. Wollstein: Jour. Med. Research, 1903, x, ii.

In 1903, Dunn[27] examined sixty-one cases at the Infants' Hospital in Boston and reported thirteen positive cases, or 22 per cent.

Weaver, Tunnicliff, Heinemann and Michael[28] studied 102 cases in Chicago in the summer of 1904 and reported twenty-six, or 25 per cent., positive for the dysentery organisms. Twenty-four of the twenty-six cases were in children over 1 year of age. All of the organisms were of the "acid" variety, the "Flexner" occurring eleven times and the "Y" type forty-seven times. These colonies agglutinated most irregularly all four types of sera.

During the same summer an extensive investigation was made in Baltimore by Knox and Schorer,[29] the full reports of which have only recently been published. In all seventy-four cases were studied and in fifty-three, or 71.6 per cent., some type of dysentery bacillus was found. In seventeen, or 22 per cent., however, it was necessary to scrape the large bowel before obtaining positive results, and so the organism was only isolated from the stool in about 50 per cent. of the cases. Shiga organisms were found in fourteen cases, and in thirteen cases mixed infection occurred.

Lucas, Fitzgerald and Schorer[30] reported thirty-eight positive cases out of forty-five cases of "infectious diarrhea" at the Boston Floating Hospital in 1910.

In England, Morgan[31] took up the study of summer diarrheas of infancy in 1905 and 1906. During the first summer Morgan examined the stools in fifty-eight cases of what he terms "acute infectious diarrhea," and twenty control cases. An organism which he calls **1 Bacillus* was found in twenty-eight of the diarrheal cases and in only one of the controls. This organism was pathogenic for rabbits and monkeys and culturally resembled the hog cholera organism of MacFadyean. The following summer he found the same organism in nineteen of fifty-four cases of diarrhea studied. He reports that in none of the cases were true Shiga or Flexner organisms found. In 1910 Williams, Murry and Rundle[32] encountered two organisms in thirty-three out of forty cases of "infectious diarrhea" in Liverpool. One, which they called *Bacillus "F,"* is closely related to the paratyphoid organism, and the other they determined to be *B. suipestifer.* Stools from 100 normal school children were examined with negative results.

The French reports of bacteriologic studies of this subject go from one extreme to the other. For example, Auché and Campagna[33] report

27. Dunn: Am. Med.; 1904, vii, 737.
28. Weaver, Tunnicliff, Heinemann and Michael; Jour. Infect. Dis., 1905, ii, 70.
29. Knox and Schorer: Johns Hopkins Hosp. Rep., 1910, xv, 1.
30. Lucas, Fitzgerald and Schorer: Jour. Am. Med. Assn., 1910, liv, 441.
31. Morgan: Brit. Med. Jour., 1906, i, 908; 1907, ii, 17.
32. Williams, Murry and Rundle: Lancet, London, 1910, ii, 730.
33. Auché and Campagna: Arch. d. méd. d. enf., 1906, ix, 513.

thirty-three cases in which the "Shiga" variety was found fourteen times, the "Flexner" seventeen and the "Strong" seven times. On the other hand, Manicatide[34] examined fifty-four cases which were selected for their resemblance to true dysentery and was able to find the organism in but five cases, all of which were in children over 5 years of age. Forty-one cases under 1 year were negative. Dumas and Menard[35] were unable to find the organism in eight patients from whom blood cultures were taken.

The study of the stools of infants with diarrhea for the purpose of ascertaining the presence of dysentery organisms has received but little attention from German pediatricians who are inclined to attribute the pathologic conditions to chemical changes in the intestine and to altered metabolism. While admitting the possibility of infection by bacteria, they believe it to be a rare occurrence and of little importance. It may be emphasized, however, that the picture of infantile dysentery with blood in the stools, much mucus and occasional false membrane is a rare one in Germany. Charlton and Jehle[36] studied sixty cases in Vienna and found the Shiga type of dysentery bacillus nine times and the Flexner type thirteen times. The cases in which the Shiga type were found were clinically more severe than those in which the Flexner organisms were found, and the stools contained both blood and false membrane. For this reason they looked on the infection with the Flexner variety as secondary or terminal. Jehle[37] further reported nine cases in which the Flexner organisms were present, and found in these cases that blood-serum also agglutinated the colon bacillus. Leiner[38] reported several cases of infection with the Flexner organism in children from 2 to 6 years of age.

The most extensive study of the anatomic lesions in cases associated with the presence of the *B. dysenteriæ* is that of Howland,[25] who reported the autopsy findings in thirty-two cases. In twenty-five of these cases the organism was recovered from the stools and in the remainder from scrapings of the mucosa at the autopsy. In five cases a pseudomembrane was present, which corresponds with one of the most frequent changes found in the epidemic dysentery of adults. Four cases showed a lymphoid hyperplasia with follicular ulceration, resembling the lesions described by Escherich[10] as "follicular enteritis," and five cases showed a superficial ulceration of the mucosa not limited to the follicles. In far the larger group of cases—fourteen, or nearly one-half—few anatomic lesions were found, and none of any importance. This group of cases included a number of badly nourished and marantic infants and several cases of terminal infection. Knox[25] reported five cases with no distinctive or

34. Manicatide: Compt. rend. Soc. de biol., 1908, lxv, 525.
35. Dumas and Menard: Compt. rend. Soc. de biol., 1907, lxiii, 601.
36. Charlton and Jehle: Tr. Assn. Am. Phys., 1904, xix, 405.
37. Jehle: Munchen. Med. Wchnschr., 1906, liii, 101.
38. Leiner: Wien. klin. Wchnschr., 1904, xvii, 695.

characteristic lesions. More recently Knox and Schorer[29] reported a case (293) in which a diphtheritic membrane was present from which almost pure cultures of *B. dysenteriæ* were obtained, but in another case (79) in the same report no dysentery organisms could be isolated from the pseudomembrane which was present. We have mentioned above the work of Charlton and Jehle[36] who found severe lesions associated with Shiga infections.

The organisms have never been recovered from the blood of an infant with ileocolitis despite the numerous attempts that have been made. By the use of the endomedium this has recently been accomplished in the case of an adult by Darling and Bates[39] and opens up a line of work which might be productive of some very important results.

A number of attempts have been made to isolate the organism from the stools of normal infants. Duval and Bassett[25] examined a series of twenty-five cases with negative results although later Duval[25] reported that he had found the Flexner organism in the stools of two normal infants. Wollstein[25] reported negative findings in thirty children, but found the organisms in the stools of three of twenty-four infants who died of other conditions. Charlton and Jehle[36] found the Flexner organism in two of ten patients examined. Collins[40] made a rather extensive study of fifty-six normal children. Three of these showed different types of the paradysentery group. She was unable to obtain the organism from the stool examinations in twenty-one cases of acute or subacute diarrhea. Kendall[25] recovered the dysentery organism in two cases of tuberculous enteritis, and Knox and Schorer[29] from a third case.

AUTHOR'S STUDY

During the summer of 1911 the opportunity presented itself to take up this question, which had not been studied to any extent since the introduction of media which permit of rapid and more accurate isolation of the dysentery organisms from the stools.

The material for the study consisted of eighty cases of acute intestinal disturbance, selected in large part from the Babies' Hospital of Philadelphia, and the remainder from St. Christopher's Hospital for Children. In addition, a number of cases from other sources were examined, but in the report we have confined ourselves to the eighty cases, which were all seen clinically by one of us (B. S. V.), and of which we have complete clinical, as well as bacteriologic records. The cases not on our own service were from the services of Drs. Sinclair, Judson and LeBoutillier at the Babies' Hospital, and Dr. Fife at St. Christopher's Hospital.

39. Darling and Bates: Am. Jour. Med. Sc., 1912, cxliii, 36.
40. Collins: Jour. Infect. Dis., 1905, ii, 620.

The cases were selected cases of a severe character and clinically were designated as ileocolitis. In every case mucus was more or less constantly present in the stools and frequently the stools were blood-streaked. It is in this type of case that the dysentery organisms have been most frequently found.

As regards age, 75 per cent. were between the ages of 3 and 8 months, inclusive. Only five were over 1 year of age. They may be roughly divided into two gross types: (1) those with acute attacks when in an apparently normal state of nutrition, and (2) those in whom there was an obvious chronic nutritional disturbance on which the acute attack was superimposed. The cases on this basis may be grouped, as regards their outcomes, as follows:

	Acute cases	Acute on chronic
Cured	19	9
Removed improved	1	2
Removed unimproved	9	1
Died	22	17
Unfavorable result	50%	62%

We have used the word "cured" in the sense it usually appears in hospital reports. It would be far better to use the word "improved," as an investigation of patients "discharged as cured" from St. Christopher's and the Babies' hospitals the previous summer has shown that approximately 50 per cent. have died subsequently from a second acute attack, or from a chronic disturbance that has followed in the wake of the first acute attack.

It is of no advantage to go into the detailed histories of the cases. They represent a group of infants who had been acutely ill with diarrhea from one day to two or three weeks before admission (average one week) and who showed frequent mucus stools on admission, with blood present in about 20 per cent. Vomiting was frequently present on admission, and was practically never absent in the history. Fever was an irregular symptom, usually present on admission, falling in the majority of cases a few days later, when it became irregular. In most of the infants living over ten days there was a tendency toward subnormal temperatures. The feeding of the infant at the time of the acute onset is tabulated as follows:

Milk (market) (diluted or whole)	24
Condensed milk	25
Modified milk (pasteurized)	9
Breast fed	10
Patent foods	1
Oatmeal water	1
Table food	7
Unknown	3
	80

The large number of infants who had been previously fed on condensed milk — a food rich in carbohydrate — is of interest in connection with Kendall's theories, which will be discussed later.

BACTERIOLOGIC EXAMINATION

The stools were collected in sterile anal tubes in the forenoon and taken directly to the laboratory where the various media were at once inoculated. One hundred and sixty-seven examinations of stools from the eighty patients were made, which were divided as follows:

No. of Cases	No. of Times Examined	Total	No. of Cases	No. of Times Examined	Total
40	1	40	1	8	8
18	2	36	4	6	24
12	3	36	—		—
2	4	8	80		167
3	5	15			

Omitting the organisms constantly present in normal stools, and considering the bacteriologic findings as a whole, the results were as follows:

Organism	No. of Cases	Percentage
B. dysenteriæ (acid)	18	22.5
Streptococcus	65	81.
Colon*	75	93.7
Pyocyaneus	24	30
The following combinations were encountered:		
Dysentery and colon		3
Dysentery and streptococcus		2
Dysentery and streptococcus and colon		13
Streptococcus and colon		50

The percentage of cases in which we found the dysentery organism is low in comparison with the majority of previous investigations. In our studies the enriching Endo-medium was used and hence but few plates were made from the stools, and it is possible that had a large number of plates been made, we would have occasionally encountered a colony of dysentery organisms in some of the cases reported as negative. In all of the cases the organisms were present in sufficient quantities to be considered as one of the predominating types of the intestinal flora. No attempt was made to subdivide the paradysentery organisms into their subvarieties, as this, as well as the serum reactions, have little if any practical importance in the light of previous studies.

Considering first the dysentery organisms the question arises as to the rôle played by them in the production of the acute intestinal diseases of infancy and early childhood. We have omitted from the paper a discussion of the clinical types of cases in which the organisms have been found. Although most frequently found in cases of ileocolitis, it is by no means confined to these cases, and has been isolated from very mild cases

* The colon organisms are enumerated because of their bearing on Kendall's studies, which are discussed in the latter portion of the paper.

of enteritis. A study of our own cases from the clinical standpoint failed to show any symptoms or combination of symptoms whereby it was possible to separate out the cases in which the dysentery bacilli were found. It has been quite definitely established that both the Shiga and Flexner types of dysentery organisms may produce dysentery, the former — in adults at least — being usually associated with a more severe clinical form, although this distinction is less clear in infants with their tendency toward gastro-intestinal disturbances. We find in infants a certain number of cases in which the stools contain large numbers of the dysentery bacilli and in some of which typical dysenteric lesions have been found at autopsy. Unfortunately we know little of the habits or method of transmission of this organism, but it has been found in the stools of apparently normal infants and in the stools of children suffering from other conditions. The earlier investigators with a few exceptions tended to blame a very large percentage of the severer intestinal cases on the dysentery organism, but a critical study shows that in a large number of their cases the organism was only occasionally found, and then with difficulty, and was of little numerical importance. The mere fact that an occasional dysentery organism is found in the stool of a case of enteritis is by no means proof that the organisms are the cause of the condition, and there is no evidence to show that in these cases the dysentery organisms have anything more than a casual relation. It seems to us that we have no right to group these cases together with those in which large numbers of dysentery bacilli are present and in which distinctive anatomical lesions are found in the fatal cases at autopsy. From a study of previous investigations and our own work we think that not more than 20 per cent. of the cases of ileocolitis may be attributed to the dysentery organisms and this figure may be high. The well known clinical fact that many of the mild cases of intestinal disturbance may pass into a severe form of ileocolitis raises the question as to whether we must consider the organism of primary or secondary importance. That is, whether a primary infection with the organism occur, producing the condition, or whether an invasion of the organism occurs after an intestinal disturbance has been produced in some manner not necessarily bacterial in nature. The answer to this is and must be merely a matter of opinion. We believe a comparatively small number are primary and that in the larger number of cases where the organism plays a rôle it is the form of a secondary invader. Our reasons for this are, first, that it has been found in all types of clinical cases and although more commonly found in ileocolitis it is typical of none; second, that in but few of the positive cases the characteristic and distinctive lesions of dysentery are found at autopsy, and third, that the organism has been found as a terminal infection in cases not primarily enteric in nature. Many objections may be raised against

this conclusion, but, as stated before, any view on this point is largely a matter of opinion.

Since the discovery of the dysentery bacillus there has been a tendency to relegate the streptococcus and other organisms to a place of secondary importance. Many of the earlier writers[6, 9, 10] considered the streptococcus of primary etiologic importance, but the value of their observations was lessened with the discovery of the dysentery bacillus, as there is no means of knowing how many of their cases were in reality dysentery infections. But few of the investigations since have taken the streptococcus into consideration, as they have been chiefly concerned with determining the presence or absence of dysentery bacilli. As will be seen by our table, the streptococcus was present in some 80 per cent. of our cases — usually in association with other organisms. In some of the severe cases, however, almost pure cultures of streptococci were obtained from the stools and the dysentery organisms were not associated in these cases. Knox and Schorer[29] reported several cases in which the streptococcus was apparently at fault and Kendall[41] has had a similar experience. While in by far the larger number of cases in which streptococci are found, the relationship is apparently nothing more than casual, in a certain number of cases it is probable that these organisms are of etiologic importance—cases in which distinct ulceration and false membrane are present in the intestine, and from which almost pure cultures of streptococci are obtained on bacteriologic examination. Too little work has been done on the other organisms, as *B. pyocyaneus,* to determine their exact importance although it does not seem probable that these, as the colon organism, are harmful in the intestinal tract.

SUMMARY

Our conclusions may be summarized at this point and are as follows:

1. The mere presence of a few dysentery organisms in a case of acute intestinal disturbance in an infant does not in itself prove that the organisms have any etiologic relation to the condition. It is only in the severe cases of ileocolitis that the dysentery organisms are present in the stools as one of the predominating types of organisms and it is these cases which show distinctive lesions of dysentery at autopsy.

2. In only about 20 per cent. of the cases of ileocolitis occurring in infancy are dysentery organisms found. While in some of these cases the infection with dysentery organisms is primary, in others it is probably secondary.

3. The cases of ileocolitis in which dysentery organisms are present cannot be separated clinically from those in which they are absent.

41. Kendall: Boston Med. and Surg. Jour., 1910, clxiii, 332; Boston Med. and Surg. Jour., 1910, clxiii, 398; Jour. Am. Med. Assn., 1911, lvi, 1084; Jour. Med. Research, 1911, xxv, 117.

4. Streptococci are present in the stools of about 80 per cent. of the cases of ileocolitis. While in the majority of these cases the relation is unimportant, it is probable that in some instances the organisms have a distinct relationship in the etiology of the condition.

Recently the study of the intestinal flora has been taken up by Kendall[11] from another view-point, namely, the "dynamic." Kendall has found that the intestinal flora of "bacillary dysentery" — a term which is loose and indefinite and which means clinically almost any type of diarrhea in its application to infants—consists of what he terms a "putrefactive group" of organisms. This is essentially a combination of *B. dysenteriæ, B. coli* and the streptococcus. He has found by experiments *in vitro* that these organisms produce putrefactive decomposition of certain sugar-free media. If sugar is added to the media he finds that the production of fermentative substances inhibits the growth of the putrefactive types of organisms and the production of putrefactive substances. From this Kendall is led to advocate the use of a sugar solution—lactose—in the treatment of "bacillary dysentery" in order to inhibit putrefaction, and he claims to have observed the disappearance of a "putrefactive" flora and the development of a normal "fermentative" group of organisms (*B. bifidus* and *B. acidophilis*) under the influence of a sugar diet.

In connection with our studies we have followed the set of bacteriologic procedures which Kendall has employed in the examination of the stools and which is designed for the use of clinicians. They are, however, too elaborate to ever become of use to workers who have not had considerable laboratory experience and access to a well equipped bacteriologic laboratory, and in addition consume too much time to be of service to one engaged in clinical work. The method of study of each stool was as follows:

1. Study of types and numbers of bacteria in gram-stained fields of freshly-passed feces.

2. Type of reaction in milk heated to 80 C. (presence or absence of gas bacilli).

3. Study of gas volume, growth and sediments in dextrose and lactose fermentation tubes.

4. Character and amount of growth in acetic acid — dextrose broth.

5. Character of colonies developing in the modified "Endo-medium."

6. Character of growth in gelatin after three days at room temperature.

7. Plating of growths on litmus-mannite-agar for identification of the dysentery organism.

The gram-stained fields were negative in by far the larger number of cases. The number of organisms varied considerably, depending on

the fecal content of the stool. We cannot say that we learned much of value from a comparison of the gram-stained fields, nor did they serve as an index to the character of the individual case. At times negative and positive fields alternated without any apparent relation to the clinical conditions or diet.

Out of nearly 100 cases examined during the summer we found but one with the *Bacillus aerogenes capsulatus* and developing a growth in milk heated to 80 C. This child was placed on raw buttermilk, but died three days later and is not included among the eighty patients reported on.

The gas formation, growth and sediments in the dextrose and lactose fermentation tubes varied to such a marked degree in different cases showing the same types of predominating organisms and in individual cases without apparent relation to the clinical condition or changes, that in reality this part of the study gave very little useful information. The same was the case with the growth on and liquefaction of gelatin plates.

A few stool reports will show the degree of variation encountered:

Case Predominating Organisms	Gas Formed in Lactose, mm.	Gas Formed in Dextrose, mm.	Gram-fields
J. G. Dysentery and colon	13	13	..
Ed. R. Dysentery, streptococcus and colon	4	20	..
B. W. Dysentery, streptococcus	15	7	..
Est. C. Dysentery, streptococcus and colon	14	31	..
J. W. Streptococcus and colon	19	20	..
H. B. Streptococcus and colon	6	2	..
L. B. Streptococcus and colon	9	1	..
Ida B. Streptococcus and colon	Tr.	15	..
Wm. B. Streptococcus and colon	23	17	..
J. B. (Lactose diet)—			
8-3 Streptococcus and colon	18	12	±
8-7 Streptococcus and colon	10	2	—
8-8 Colon and subtiloid	20	8	±

Unfavorable results in 58 per cent.

We found the modified Endo-medium most satisfactory for separating out the dysentery organisms in the stool and a distinct advance over previous methods.

In so far as the bacterial picture of ileocolitis consists to a large degree of some of the "putrefactive group" of organisms, i. e., combinations of *B. dysenteriæ, B. coli* and the streptococcus, our findings were in accordance with those of Kendall. In thirty-one of the eighty cases a "lactose diet" was used despite certain theoretical objections, and the results were as follows:

Removed improved	1
Removed unimproved	4
Cured	12
Died	14
	31

The results were much the same as in the whole series of eighty cases, but are too few in number to draw very definite conclusions. In those cases which were receiving lactose and improving, the "putrefactive" organisms gradually disappeared as the predominating types of organisms in the bacteriologic picture, while in those cases not improving they persisted. In other words, the disappearance was apparently dependent on the condition of the patient rather than on the diet.

While we have no argument as to the *"in vitro"* experiments of Kendall, several objections may be raised against his ideas as to the production of the acute intestinal diseases in infancy. According to his views the action of the "putrefactive group" of organisms is to produce putrefactive decomposition in the intestine and the products of this decomposition are the cause of the intestinal disturbance. So far as we know it has never been shown that the products of putrefactive decomposition arising from the action of *B. dysenteriæ, B. coli* and the streptococcus produce the intestinal disturbance, and in this respect one of Kendall's chief premises is an assumption. Clinical evidence and experience go to show that in many instances the fermentative products are at fault, and that carbohydrates are more apt to produce trouble than any other single food element. The use of a food with a high protein content and with a low carbohydrate percentage is frequently followed by excellent results. If Kendall's treatment is to be used in those cases alone which show *B. dysenteriæ* in the stools, it is of little value, as these cases cannot be separated clinically, and to wait until a stool examination has been made would be to waste considerable time which might in many cases determine the outcome of the case.

USE OF POLYCARBOHYDRATES IN THE DIET OF THE YOUNG INFANT *

JULES M. BRADY, B.S., M.D.
Assistant Professor Diseases of Children St. Louis University
ST. LOUIS

In recent times the rôle of the carbohydrates in infant feeding has received marked attention. Whether the importance they have assumed in the minds of some in the pathology of nutritional disturbances is justified must be decided by future study.

For years the adjustment of the carbohydrate percentage to the digestive and caloric requirements of the infant was considered the easiest part of the problem of infant feeding. The fact that mother's milk contains constantly 7 per cent. of lactose, furthermore the fact that lactose is of animal origin and is essentially the same whether human or derived from the cow, ass, rabbit, dog or horse, made this portion of the problem seem simple. To imitate Nature and furnish the infant with the same carbohydrate in the same percentage appeared to answer every requirement.

That lactose could cause digestive disturbances with the production of acid, green, foamy, watery stools as a result of fermentation has been recognized for many years. This disturbance was not considered of special frequency nor of great importance.

Many of our leading pediatricians hold lactose to be the ideal sugar for an infant; furthermore, the addition of a cereal to the diet of a young infant was not only considered unnecessary but actually harmful. For years it was believed that the young infant had little power of digesting starch, and, furthermore, the fact that the breast-fed baby throve on a starchless diet strengthened them in this position. The early use of starches has been considered a frequent cause of intestinal indigestion; this disturbance frequently not manifesting itself until a later period of infancy. Still[1] believes that barley water in the milk mixture of a young infant plays an important rôle in the causation of rickets. He says "the addition of starch in any form to the diet of an infant under 8 months of age is liable to interfere with assimilation and in this way involves the danger of rickets, especially if the milk mixture is

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*From St. Ann's Infant Asylum and Pediatric Department, St. Louis University.

1. Still: Common Disorders and Diseases of Childhood, 1909.

already poor in fat." Holt[2] teaches that the average healthy infant is best nourished for the first four or five months on the elements of milk. He considers the addition of the various cereals to the milk of young infants as a useful measure for some, but not desirable for all. He states that the early use of much farinaceous food often results in serious harm. Roach[3] in 1906 stated that starch should not form a part of the infant's food in the early months of the first year. Jacobi[4] has for years persistently advocated barley as a diluent with the addition of cane sugar. While not overlooking the nutrient effect of the barley he believed the mechanical effect on the proteins was of great importance.

The work of Finkelstein and his pupils has caused the carbohydrates to receive the attention they deserve. That lactose may be the immediate cause of dangerous symptoms of intoxication in some infants, particularly if there has been an injury to the intestinal epithelium, is now admitted by many. Recently Leopold[5] has advised a combination of dextrin and maltose in about equal parts as the best form of carbohydrates for the young infant.

Czerny[6] selects milk-sugar for use during the first months of life and avoids the employment of other sugars or carbohydrates during the early weeks. Of lactose he adds 1 teaspoonful to each of five feedings in twenty-four hours. From the fourth month instead of increasing the amount of sugar he adds flour. He thinks by the early and systematic use of flour the danger of rickets and scrofula is not increased. He employs oat flour, wheat flour and cornstarch and considers them of more value than dextrinous flours. He believes the favorable influence of flour is due to the slow saccharification, which brings about a slow absorption of water, due to the sugar, which is more readily compensated for by the organism.

In a service of six years in one of the largest infant asylums of the West we have put into practice every possible method of infant feeding that has seemed justified. Mixtures of cow's milk and lactose, with the percentage of protein, fat and carbohydrate corresponding to the generally accepted principles of infant feeding have been employed innumerable times, but leave much to be desired. In recent times the results in our institution approach those obtained in private practice. Our results in the past compare very favorably with those reported by Mr. Homer Folks,[7] secretary of the New York State Charities Aid Association, who in 1910

2. Holt: Diseases of Infancy and Childhood, 1911.
3. Roteh: Pediatrics, 1906.
4. Jacobi: Therapeutics of Infancy and Childhood, 1903.
5. Leopold: Arch. Pediat., xxviii, No. 10, p. 844.
6. Czerny: Des Kindes Ernährung, Ernährungs-Störung und Ernährungs-Therapie, 1909.
7. Folks, H.: Bull. Acad. Med., N. Y., February, 1910.

through the kindness of the Children's Department of the Russell Sage Foundation, investigated the mortality statistics of the infant asylums of the country. All of us recognize the difficulties under which we labor. The overcrowding, the lack of help and the frequency of epidemics all conspire to thwart our efforts. So great have the difficulties seemed that many have seriously considered the advisability of doing away with infant asylums. For many reasons such a course is not practical.

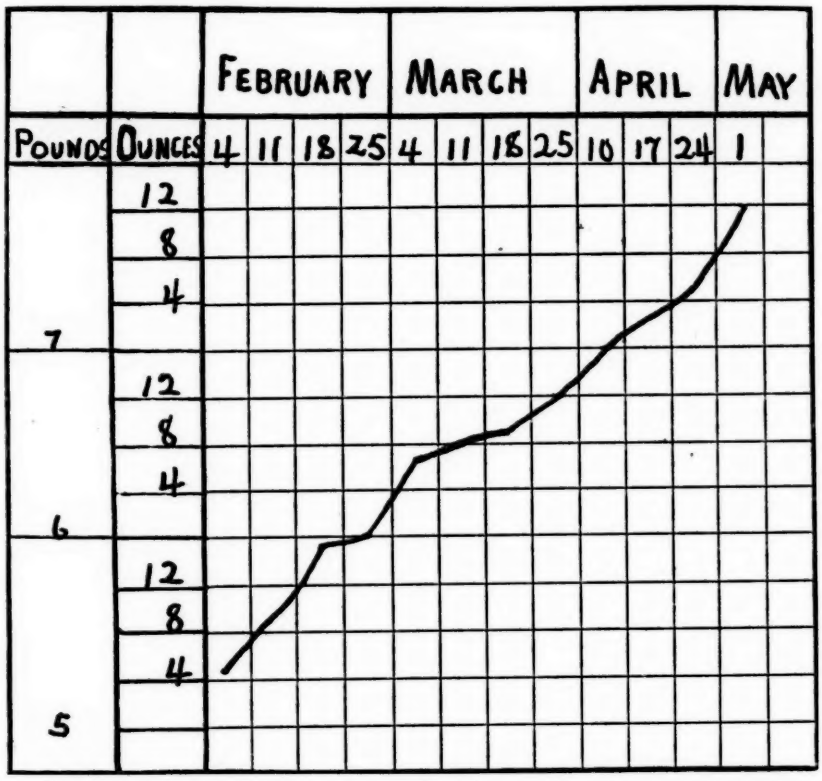

Chart showing weight curve of a much under-weight asylum infant receiving the polycarbohydrate diet.

The usual milk modifications employed so successfully in private practice do not give results in asylums. The asylum infant is a special problem, and in order to promote nutrition extraordinary measures must be resorted to. We have found that these infants are greatly assisted in battling with the surroundings by the use of a variety of easily assimilable carbohydrates. A high percentage of protein is fed even to the new-born infant and the percentage of fat is closely watched. Beginning with a fat-free diet at birth the percentage is gradually raised with the increase in weight of the baby. With the addition of fat to the mixture,

owing to its high heat value, the twenty-four-hour amount must be reduced so as not to exceed the caloric requirements of the infant.

MILK MIXTURE

One and one-half ounces by measure of barley flour is cooked with 16 ounces of water at least twenty minutes down to 10 ounces, and added to 20 ounces of skim sweet or acidified milk.[8] The latter we believe for many reasons is preferable in an institution. One-half ounce by measure of cane sugar and 1 ounce by measure of malted food containing dextrin and maltose are added. The carbohydrate content of the mixture would be represented by lactose 2.66, cane sugar 1.30, maltose and dextrin, 1.70, plus barley, 2.25, which is partly dextrinized. This gives a total carbohydrate percentage of 7.91. One liter has a caloric value of 415.

We have used this mixture over a period of eighteen months. Last July, owing to the favorable results, it was adopted as the routine formula for new-born infants. We do not wish to be understood as believing that the problem of infant feeding is going to be solved by one mixture that need never be varied. The individual infant will always furnish the indications for what is best for its metabolism. In an infant asylum, owing to the large number of babies and the scarcity of help, the best results will be obtained by minimizing the work and prescribing a few formulas which give the best results for the average infant. We are satisfied of the wisdom of supplying the young infant a liberal percentage of protein as represented in this mixture. This percentage we do not change throughout the first year; the same may be said in regard to this mixture of the various carbohydrates. The fat percentage demands a change from time to time; beginning with a fat-free diet, the percentage is gradually raised with the progress of the baby.

CLINICAL RESULTS

Over a period of eleven months this mixture was adopted as a routine formula in the ward for the young infants. One hundred and seventy babies were cared for; ninety babies received breast milk for the first two weeks of life, and then received this mixture. Forty-five received this mixture from birth; eighteen were between 2 and 3 months of age and seventeen, owing to nutritional disturbances, were on *Eiweissmilch;* these received this mixture after completion of the *Eiweissmilch* cure. During this period there was a remarkable absence of babies with nutritional disturbances; the stools were uniformly of good color and consistency. No infant on this mixture in this series developed the symptom-complex described by Finkelstein under the caption alimentary intoxication,[9] nor did symptoms arise which could be interpreted as *Mehlnahrschaden* (starch injury) in the sense of Czerny and Keller. Twelve babies did not show a satisfactory gain, so they were transferred to

8. Brady, J. M.: Arch. Pediat., 1910, xxvii, No. 6.
9. Finkelstein, H.: Jahrb. f. Kinderh., January, 1907, p. 1.

Eiweissmilch. All the other babies throve satisfactorily, the fat percentage alone requiring alteration. Eighteen babies died, a mortality of 10.5 per cent. The causes of deaths were as follows: two sudden deaths, nutrition good; autopsy showed enlarged thymus; one, premature birth; one, congenital syphilis; seven, acute bronchopneumonia, nutrition good; one, phlegmon of the scalp; and six, decomposition (Finkelstein).

It will be seen from the above that 170 babies all under 3 months of age were cared for with only six deaths from nutritional disturbances. It is the experience of all that the great difficulty in an asylum is in establishing the nutrition of the infants. In passing it is to be remarked that while it is the desire to show the favorable results of this polycarbohydrate diet, it must be emphasized that the use of *Eiweissmilch* has prevented the deaths of a number of babies. The routine adoption of this diet for the young infants has limited very much the use of *Eiweissmilch*. Our infants remained under observation for variable periods ranging from one to six months; the large majority for a longer time than three months. I recall that the death-rate during the first year in private homes is generally stated to be 20 per cent.; Cautley says infant mortality is very high in the first month, sinking to less than half in the second month, and progressively decreasing with each successive month during the first year.

Statistics frequently deceive and therefore I believe just as much value may be placed on the impressions of the observer; I therefore offer the above figures for what they are worth.

DISCUSSION

In the past we have erred by feeding too much fat and too little protein and have condemned the use of cereals. The typhoid fever patient of to-day is assisted through his illness by a more liberal use of foodstuffs; likewise, the asylum baby can only withstand the results to which it is subjected by a liberal diet. Formerly we believed that the young infant could not digest starch; that it did not thrive owing to the fact that the digestive organs were not properly developed. Now we know that the new-born infant is abundantly supplied with all the digestive ferments. Diastatic ferment is found in the parotid gland as early as the fourth month of fetal life. There is no dearth in the supply of lactase, maltase, saccharase and amylolytic ferments in the infantile alimentary tract; therefore the ability to care for a variety of carbohydrates. Why the metabolic processes are favored, leading to an increased storage of salts and water in the tissues, is not apparent, but clinical results prove this. The very best indication that an infant is thriving is the presence of a regular gain in weight; if this does not occur the clinician cannot remain passive; something must be done. A sudden increase of weight

such as accompanies the development of edema, due to a pathologic state of the endothelial cells lining the capillaries, should not be misinterpreted. Use of these various carbohydrates in the dietary of the young infant with the careful exhibition of the fats seem to have a strong influence in maintaining the normal salt balance which we now recognize is a *sine qua non* in the metabolism of the healthy infant.

That the nutrition of an infant is favorably influenced by the use of more than one carbohydrate has been emphasized by Keller[6] and Czerny, who speak of the use of two carbohydrates, either wheat flour and cane sugar, or wheat flour and maltose, in case of disturbed nutrition.

Striking results have been met with in infants in private practice who have failed to thrive and have lost weight. It is a mixture well adapted to follow up a malt soup[10] or *Eiweissmilch* cure.

It has been determined that normally 90 per cent. of the heat requirements of the infantile organism are furnished by the sugars and fats; for a time a deficiency of the one may be compensated for by an increase of the other without ill effects. That fat in the diet of some, particularly the marantic and asylum infant, tends to have a *decomponirend* action, makes it advisable in the early weeks to allow the carbohydrates to furnish the energy usually supplied by the fats until the weight has started upward. This one-sided diet for well-known reasons must not be continued over too protracted a period.

Metabolism experiments show that the protein in the diet of the normal infant should supply not more than 10 per cent. of the total energy in the food. These experiments indicate that when these figures are exceeded there is danger of its specific dynamic action, whereby the total heat production of the body is markedly elevated. Not only does this lead to fever, but the digestive tract is enormously taxed, likewise the kidneys. That the intermediary metabolism is unfavorably influenced is also claimed.

One liter of the above mixture contains 23.11 gm. of protein, which, multiplied by 4.1, the heat value of 1 gm., equals 94.75 calories. To cover its caloric requirements an infant weighing 8 pounds and 13 ounces would require 1 liter equaling an energy quotient of 100. In this diet the protein would furnish the infant 22.83 per cent. of the total energy of the mixture. Theoretically for the above-mentioned reasons this would be very undesirable. Practically, for the asylum infant and the marantic infant in private practice, it does not apply. These infants thrive on this high protein diet in association with the liberal use of the various carbohydrates. Any unfavorable effect from the proteins exerting their specific dynamic action is not apparent clinically. We have repeatedly attempted to advance the nutrition of our infants by feeding a protein

10. Brady, J.: Jour. Am. Med. Assn., March 16, 1912.

percentage which would not furnish more than 10 per cent. of the total energy of the food. Results far superior were obtained with the percentage indicated.

That the above statement that the proteins should not furnish more than 10 per cent. of the total energy of the food does not hold in practice for the run-down infant is shown by the results obtained by feeding *Eiweissmilch,* 1 liter of which without the addition of sugar contains 417 calories; 4 per cent. addition of a malted food raises the caloric value to 550. The mixture contains 30 gm. of protein to the liter which, multiplied by 4.1, yields a caloric value of 123. This would mean that the protein in *Eiweissmilch* as usually fed would represent 22.36 per cent. of the total energy in the food.

Metabolism experiments indicate that flour in the diet such as barley causes an increased retention and utilization of nitrogen; protein is stored in the body with a resulting increase in weight and growth.

RICKETS

The greatest objection will be raised by those who see a close relationship between starch in the diet and rickets. This belief is founded on clinical observations. The cause of rickets is unknown and according to Zappert[11] of Vienna no more is known to-day about the nature of this disease than was known fifty years ago. That diet must play an important rôle in its causation is shown by the scarcity of severe forms of the disease amongst the breast-fed.

When we see in an asylum infant after infant fed on milk diluted with water with the addition of milk-sugar, fail little by little until at 6 weeks or 2 months of age it remains but a shadow of itself, while on the other hand we visit a ward of seventy infants and find the large majority of these infants making regular weekly gains, who would not declare for the diet of the latter no matter how much at variance it seems to be with the natural food?

If we consider the prominences of the epiphyseal boundary of the ribs with flexibility of the cranial bones at the margin of the sutures evidence of rickets, then 80 per cent. of our infants will show the disease. But that many of our infants show the condition in a severe form we cannot admit. Kassowitz of Vienna, Epstein of Prague and Kissel of Moscow calculate that 89 to 90 per cent. of the children of their out-patient clinic are rachitic. Then again, there are authorities such as Fischl, of Munich, who among others found only 5 per cent. of their clinical material rachitic. The difference in these figures must be due to the variations in views in regard to what is sufficient to establish the diagnosis.

11. Jacobi: Dis. of Child., 1911, p. 237.

CONCLUSIONS

1. Mixtures of milk, water and lactose with fat, protein and carbohydrate in the percentage corresponding to the widely accepted principles of infant feeding do not give satisfactory results in an infant asylum.

2. The asylum infant, even in the early weeks, is greatly assisted in making gains and weathering the unfavorable surroundings by a liberal use of barley, maltose, dextrin and cane-sugar in the diet.

3. The exhibition of polycarbohydrates in the diet is an excellent therapeutic agent for the infant in private practice who refuses to gain on the usual milk mixtures or has already run down on the same.

4. For this diet to be successful the protein must be liberal in amount and special attention must be paid to the fat, which should only be raised with the increase of the weight of the baby.

5. The fear of rickets need not be considered; the first requirement is that the infant be kept alive. *"Probiren geht über studiren."* Practical experience overrides theoretical conclusions.

APPENDICITIS IN CHILDHOOD

RUSSELL S. FOWLER, M.D.

Chief Surgeon, First Division, German Hospital of Brooklyn; Surgeon, Methodist Episcopal Hospital, etc.

BROOKLYN, N. Y.

There have been cared for in the first surgical service at the German Hospital of Brooklyn (1900-1912) 183 patients with appendicitis under the age of 12 years. Two patients, moribund on admission, were not operated on and died. In two interval cases the patients were operated on and recovered. The remaining 179 were acute cases in which the patients were operated on, of whom twenty died.

Proportion of Children to Adult Cases.—The total number of cases during the same period was 1,115, the proportion of children (183) 16.41 per cent.

The present report is founded on the last hundred consecutive personal operations for acute appendicitis in patients under 12 years of age.

Mortality.—Three of these patients died in the hospital; one patient with abscess died months later following resection of the intestine for tuberculosis and obstruction. Two of the three deaths from operation were in diffuse septic peritonitis cases, the other in a case in which a very acutely inflamed appendix with a perforation, operated on in less than twenty-four hours, and with an escaped enterolith which lay post-cecal directly on the iliac vein. This patient died in forty-eight hours of septicemia in spite of vaccine therapy and supportive measures. The operative mortality was 3 per cent.; the disease mortality 4 per cent. There has been but one death in the last fifty-six cases. (In the last hundred personal operations for acute appendicitis in adults six died.)

Age.—The youngest patient was 2 years and 9 months old; the oldest 12 years.

Age, Years.	No. of Cases.	Age, Years.	No. of Cases.
11-12	19	6-7	9
10-11	18	5-6	5
9-10	13	4-5	6
8- 9	14	3-4	2
7- 8	13	2-3	1

Sex.—Sixty-three were males; thirty-seven females.

Previous Attacks.—Eleven gave a history of one previous attack; four had two previous attacks; two had three previous attacks; one had four previous attacks; eighty-two patients were operated on in the first attack.

Duration of Attack.—Nineteen were operated on in the first twenty-four hours; thirty-three in the second twenty-four hours; twelve in the third; twenty-one in the fourth; four in the fifth; seven on the seventh day; four after two weeks.

Blood Examination.—In seventy cases the examination of the blood was recorded. The average leukocyte count was 19,106; the average polynuclear, 79.7 per cent. The highest leukocyte count was 48,200; the lowest, 8,200; the highest polynuclear, 92 per cent.; the lowest, 63 per cent.

Condition Found at Operation.—Inflammation limited to the appendix (in this class are included cases of all degrees of severity of inflammation, perforation and gangrene, so long as there is but slight involvement of the peritoneum in the neighborhood), thirty-six cases; with localized abscess, twenty-one cases; with abscess and spreading peritonitis, two cases; spreading peritonitis without abscess, eight cases; with diffuse septic peritonitis, thirty-two cases; with localized peritonitis, one case.

Cause of Death.—Two of the deaths could have been prevented by early diagnosis; two deaths were not preventable; in one there was sepsis from the appendix directly entering the circulation through the medium of the iliac vein; in the other the patient died two and a half months after the original operation as a result of a resection operation for tuberculosis with intestinal obstruction.

Operative Technic.—The appendix is larger in proportion to the rest of the intestine in childhood, while the mesentery of the appendix is not apt to be so well developed as in adult life. Removal of the appendix in all cases with as little disturbance of the neighboring intestine as possible; wick drainage if necrosis is present in the neighborhood of the appendix; glass tube drainage of the pelvis for diffuse peritonitis; wound closure except for the emergence of the drain. Vaccine therapy in septic cases.

Postoperative Treatment.—Rest in bed. Fluids 2 to 4 ounces every two hours and saline by rectum for the first twenty-four hours if no peritonitis is present. Murphy proctoclysis in all cases deprived of fluids. Deprivation of fluids by mouth if peritonitis is present until normal wound temperature is reached; elevation of the head and trunk in all peritonitis cases to lessen the rapidity of absorption; enema daily and as frequently as necessary to control distention; gastric lavage if stomach is distended.

Complications.—The complications are the same as in adults. Diffuse septic peritonitis occurs more readily in children as the omentum is thin and poorly developed and for this reason cannot afford the efficient barrier to spreading inflammation so often observed in the adult. Sec-

ondary abscesses are more common with diffuse septic peritonitis in children.

Dilatation of the Stomach.—Dilatation of the stomach from excessive fluids occurs more frequently in children than in adults. It is early and easily recognized if frequent palpation of the upper abdomen is made a routine, and quickly remedied by lavage and restricted fluids.

Diagnosis.—Nothing shows so well the advance in the diagnosis of appendicitis as a comparison of the number of cases occurring in children diagnosed during the first decade in the history of appendicitis, and the number in which the diagnosis is now made. From 1887 to 1897 at the Methodist Episcopal Hospital of Brooklyn (Drs. George R. Fowler and Lewis S. Pilcher, surgeons) there were treated 340 patients with appendicitis, of whom but twelve were under the age of 10 years; thus $3\,^{9}/_{17}$ per cent. of all cases were in children. It is noteworthy that not a single female child was treated for appendicitis in this series.

From 1900 to 1912, 1,115 patients were treated in the first surgical service at the German Hospital of Brooklyn, of which number ninety-two were under the age of 10. A comparison of these figures speaks well for the progress made by the general practitioner in diagnosis.

The History of the Case.—I am impressed with the belief that the diagnosis of appendicitis can be made in the majority of cases with a fair degree of certainty before the disease has advanced sufficiently to cause pain in the right iliac fossa. The early symptoms are sudden, gradually increasing pain, not necessarily severe, in the epigastrium or about the umbilicus, with some diffuse abdominal pain not accompanied by tenderness. If food has recently been partaken of, the stomach is emptied by vomiting. The symptoms persist for a few hours, then disappear not to return for a variable length of time. These symptoms recurring from time to time in a child and not directly assignable to other causes are the early symptoms of appendicitis. I have operated soon after these symptoms and found indubitable evidence of recent inflammation of the appendix. Usually the history is practically the same as in adults, with this exception, that whereas the average adult with an attack of cramps which later localizes in the right iliac fossa will keep quiet and permit the inflammation to localize or subside, children will be up and about as soon as the severe pain has passed.

Overfeeding and ill-advised feeding are so common in children that this factor must always be considered. It is quite frequent to get a history of extraordinary indiscretion and no doubt this acts as a causative factor in the production of the disease. If the eyes are dull and the child listless with a fever out of proportion to the abdominal symptoms, intestinal indigestion should be considered. In any event a blood examination will be of material aid. The factor of possible "indigestion" is apt to

cause delay, as the parents usually give the child a laxative when the pain is first complained of. This may result in a spread of the inflammation.

Usual History.—The child is taken suddenly ill with pain in the abdomen and coincident vomiting; a cathartic is given, following the action of which the patient is relieved and plays about, though occasionally complaining of cramping pain. The pain continuing a physician is summoned during the first twenty-four hours if the parents are intelligent, otherwise later. The diagnosis can be made in the first twenty-four hours on this history plus localized rigidity at any part of the abdomen. Fever may be absent or there will be slight fever, 100.5 and very slight, if any, acceleration of pulse, 90 to 100. In more severe cases the above symptoms are present in an exaggerated form and there is added painful distention and diffuse abdominal tenderness (spreading peritonitis).

A history of previous attacks is of value if classical; i. e., diffuse abdominal pain or pain about the navel or with subsequent localization at any point. It is also of value even though there is no history of localization if indiscretion in diet can be ruled out.

Temperature and *pulse* are of hardly any value in diagnosis except that a high temperature early in the case would rather cause one to suspect some lesion (pneumonia) other than appendicitis. Quite often the abdomen is opened in a patient with a temperature of 100 F. and a gangrenous condition of the appendix is disclosed. Taken in connection with other symptoms the temperature and pulse may be of some value in determining how the patient is combating the infection, but a low temperature must not lead to a sense of safety.

Vomiting is a common symptom but present in many of the diseases of childhood. It is of value when there is a history of several attacks of vomiting with diffuse pain or pains at the navel, without any indiscretion in diet.

Condition of the Bowels.—Usually the bowels have been moved by a cathartic before a physician is called. This practice should be discouraged as it tends to spread any existing inflammation and may transform a case with early perforation and localized peritonitis into a diffuse septic peritonitis.

The bowels are usually *constipated* at the beginning of the attack. The constipation has usually not existed for more than twenty-four hours, and there is slight distention. This distention is responsible for some of the pain, which latter is much relieved following a successful enema and the passage of gas. Paresis is a rare complication in children. If present there is *absence* of *peristalsis*. This is the differentiating symptom between paresis and mechanical obstruction and obstruction from localized peritonitis.

Diarrhea may occur but is rare. If there are blood-stained, small, repeated movements with straining, intussusception is probable.

Abdominal Examination.—In the majority of cases the diagnosis can be made by the abdominal examination, irrespective of the history. In making the examination the hands should first be warmed to avoid any abdominal reflex from cold. Tact and gentleness are necessary. If possible the confidence of the child should be secured; if not, the attention should be distracted. The posture of the patient is not of significance unless he lies on his back with the knees drawn up and recurs to that position when permitted, or when slight pressure is made over the appendix. Such a position indicates early in the disease that the appendix lies in the neighborhood of the anterior abdominal wall; later in the disease, that there is abscess formation along the iliac-psoas muscle. The patient may lie on the left side with the knees drawn up, not necessarily because there is great pain but because this is the easiest posture to assume.

For examination the patient is placed on the back, the abdomen, thighs and chest exposed and the limbs drawn down. Inspection will reveal the condition of the intestinal tract, whether distended or not, and in thin patients the presence or absence of peristaltic movements, the type of respiration, and in some instances the presence of a tumor. Respiration in young children is normally of the abdominal type; therefore if the abdominal muscles are held rigid and the costal type of respiration be present, a painful abdominal condition or a pulmonary condition will be suspected. The respiratory rate, unless excessive, is not of much value as it may be due to distention from any cause. While palpating the abdomen it is best to watch the child's face and to indulge in such conversation as will tend to distract the attention, not alone for this reason but also because a child's face is the mirror of his feelings and one can detect pain or tenderness there as the fingers gently palpate the abdomen. If successful in distracting the attention, palpation is easy, though true muscular rigidity will be harder to obtain than tumor.

Local muscular rigidity is easier to elicit than general. It is at times difficult to say whether general rigidity is voluntary or not. General rigidity may also be present in pneumonia and diaphragmatic pleurisy. Real rigidity is absent if the appendix is in the pelvis.

Tenderness is difficult to elicit as the child does not easily differentiate between pain and tenderness. Fear must be overcome to be certain.

Tumor is present or absent according to the period of the attack. In slowly progressive cases a tumor is formed in the second twenty-four hours. In mild cases and in rapidly progressive cases (cases of early perforation) there is no tumor. The advent of a tumor should not be awaited.

The loin is also palpated, as in quite a few cases the appendix is retrocecal and to the external side of the colon so that abdominal rigidity is lacking while present in the loin. In non-descent of the cecum the rigidity and tenderness is at the level of or even above the umbilicus. The appendix may stretch across the abdomen and be inflamed at the tip so that the inflammatory symptoms are then on the left side. Failure to discover rigidity and tenderness in the abdomen or loin does not rule out appendicitis, for the appendix may lie in the pelvis, in which event a rectal examination is essential.

Rectal Examination.—Delatour in 1898 called attention to the value of rectal examination in doubtful cases.[1]

Rectal examination is particularly valuable in children for the examining finger reaches much further than is the case in an adult. Its routine employment would lead to earlier diagnosis in a considerable proportion of cases, while in some cases in which the appendix lies in the true pelvis it is only per rectum that the enlarged appendix or abscess can be felt.

Anesthesia as an Aid to Diagnosis.—The abdomen of a child, owing to the thinner abdominal walls and more shallow pelvis, is much easier to palpate, having gained the child's confidence, than the abdomen of an adult. In doubtful cases, having excluded pulmonary disease and exhausted other means, an anesthetic may be administered and the abdomen palpated. I have never found this necessary, but can imagine that in certain cases it would be of value. Under anesthesia it is easy in children, except in cases with extreme distention, though not always desirable for fear of rupturing adhesions, to palpate the appendix.

Should anesthesia be used for purposes of diagnosis it would be well to have everything ready for immediate operation.

Diagnosis of Unusual Cases.—At times the diagnosis is very difficult. There are cases in which the appendix lies in the pelvis and in which the infection is pure colon bacillus. In such cases the exciting cause seems to be a marked indiscretion in diet. A physician is called to attend a child who has vomited indigestible material, for instance, a last year's candy Easter egg or a breakfast of discarded bananas, as in two of my cases, and the vomiting is frequently repeated. There is diffuse abdominal pain, distention, no rigidity, slight diffuse tenderness. The patient vomits and has diarrhea. There is slight fever sometimes or none at all. The natural diagnosis in such instances is gastro-intestinal irritation from indigestible food and the treatment usually followed is a course of calomel followed by magnesia and starvation. The bowels move, the diffuse abdominal pain disappears and the child is kept in bed for a few days. Apparently

1. Delatour: Brooklyn Med. Jour., November, 1898.

all is well when suddenly the temperature rises, there is pain in the lower abdomen and an examination shows rigidity in the lower part of the abdominal wall. It is then that the diagnosis of appendicitis is first thought of. Operation discloses an abscess in the pelvis surrounding the appendix and a condition of spreading peritonitis. Such patients usually recover though the convalescence is much prolonged and is apt to be accompanied by secondary abscess formation. In these cases an early digital exploration of the rectum would have disclosed the condition. The explanation of such cases lies in the location of the appendix and the fact that the infection is a pure colon one.

Differential Diagnosis.—Many patients are referred to the surgeon for operation for appendicitis who have not appendicitis. Some of these patients have no surgical lesion at all and are cases of pneumonia, typhoid fever, menstruation, distended bladder, fecal impaction or errors in digestion; others have surgical lesions not demanding operation, as hip-joint disease; while still others, and these are in the majority, have intussusception or other form of intestinal obstruction, abdominal tumor, tuberculous peritonitis or torsion of the ovary.

It behooves the operator to be exceedingly careful in his diagnostic methods or he will have the mortification of operating unnecessarily. A blood examination should be made whenever there is any doubt as to the diagnosis. It will rule out certain lesions with certainty. The blood count in children does not offer any peculiarities.

In typhoid fever if there is doubt the blood examination will show whether there is typhoid fever alone, or whether, as is true in a small proportion of cases, there is also an appendicitis.

301 DeKalb Avenue.

PROGRESS IN PEDIATRICS

INFANT FEEDING AND DISORDERS OF THE DIGESTIVE SYSTEM

RÉSUMÉ OF THE LITERATURE OF THE PAST SIX MONTHS

MAURICE OSTHEIMER, M.D.
PHILADELPHIA

INFANT FEEDING

Breast Feeding.—Everybody will agree with Hunt[1] that no one has ever composed a substance which can replace human milk for the well baby. During the two weeks after birth Jaschke[2] gave breast milk only to 100 institution infants. If a mother was ill or had not enough milk, another mother nursed the infant in addition to her own baby. Eighty-one gained in weight regularly. Jaschke believes that, where nursing mothers are always to be had, every infant can be breast fed successfully. Rietschel[3] fed seventy-three infants on the breast solely during the first six weeks of life. He allows but six feedings in twenty-four hours, rarely seven or even eight. Laurentius,[4] who studied twenty-one wet-nurses, found that, when one mother nurses several infants, her milk-supply is enormously increased. Helbich[5] secured similar results with the regular use of the breast-pump in ten women.

That breast milk is not always an ideal food for sick babies has been shown by Langstein and Hörde,[6] who pumped out the breasts and gave the milk to 188 institution infants, twenty-nine of whom died (15 per cent.); 111 recovered (59 per cent.), while forty-eight showed no improvement (26 per cent.). They advise pumping out the breasts and giving the milk to the baby during the first days of life, so that both breasts are emptied regularly and the baby receives enough food without too great exertion. On the other hand, Wieland[7] and Variot and Morancé[8] consider that breast milk is of therapeutic value in the nutritional disturbances of infancy. Strathy[9] states that the breast-fed baby

1. Hunt, R. B.: Boston Med. and Surg. Jour., 1912, clxvi, 367.
2. Jaschke, R. T.: Monatschr. f. Geburtsh. u. Gynäkol., 1912, xxxv, 60.
3. Rietschel, Hans: Jahrb. f. Kinderh., 1912, lxxv, 403.
4. Laurentius, Johannes: Arch. f. Kinderh., 1911, lvi, 275.
5. Helbich, H.: Monatschr. f. Kinderh., 1912, x, 311.
6. Langstein, L., and Hörde, E.: Therap. Monatsh., 1911, No. 12.
7. Wieland, Emil: Cor.-Bl. f. Schweiz. Aerzte, 1912, xlii, 149.
8. Variot and Morancé: Bull. Soc. de pédiat. de Paris, 1912, xiv, 120.
9. Strathy, G. S.: Canadian Practitioner and Review, 1912, xxxvii, 76.

is brought to the physician only because of failure to gain in weight or for colic. For the former he advises rest, exercise and correct food for the mother; for the colic he orders regular feedings at longer intervals and also reduces the time of each feeding. Lavage may be of benefit. Until such attempts have failed the baby should never be weaned; then he begins with mixed feeding.

The importance of breast feeding is the main theme of articles by Elliott,[10] Spriggs,[11] Hale,[12] Mills[13] and Labbe.[14] Davis[15] points out its value in the prevention of infantile mortality. He found that 72 per cent. of 736 babies born in Boston were breast fed. Howard[16] tells how, in the milk stations of the Boston Milk and Baby Hygiene Association, one-third to one-half of all infants are breast-fed, partially or entirely. This supervision has materially lengthened the period of breast feeding.

That infants who have been weaned may again be put back on the breast with success has been shown by Wile,[17] who reports six infants nursed again ten days to two months after weaning. Supplemental feeding may be necessary for a month after replacing the baby on the breast. After a contagious disease or breast abscess efforts should be made to get the baby back to the breast. In the case of puny infants an older, stronger child should be put to the mother's breasts to develop a supply. Mayerhofer[18] reports several similar cases.

Meigs and Marsh[19] have made a series of analyses of human milk which confirm Meigs' earlier work. The protein is about 1 per cent. and very slight changes occur during lactation. Friedenthal[20] discusses the important rôle played by the salts of human milk in the feeding of infants. Dibbelt[21] draws attention to a physiologic lack of lime salts in human milk when the infant is about 7 months old. This may be a cause of rachitis. That milk from an eclamptic mother should not be given the baby is Frost's[22] conclusion, as three died of the four infants whose mothers nursed them after convulsions. Davis[23] agrees with him,

10. Elliott, T. H.: West Virginia Med. Jour. 1912, vi, 378.
11. Spriggs, E. I.: Clin. Jour., 1911, xxxix, 33.
12. Hale, S. E.: Southern Med. Jour., 1911, iv, 821.
13. Mills, A. E.: Australian Med. Jour., 1912, ii, 428.
14. Labbe, E. J.: Northwest Med., 1911, iii, 312.
15. Davis, W. H.: Boston Med. and Surg. Jour., 1912, clxvi, 242; and Am. Jour. Pub. Health, 1912, No. 2, p. 67.
16. Howard, A. E.: Boston Med. and Surg. Jour., 1912, clxvi, 773.
17. Wile, I. S.: Jour. Am. Med. Assn., 1912, lviii, 775.
18. Mayerhofer, E.: Wien. klin. Wchnschr., 1912, xxv, 272.
19. Meigs, A. V., and Marsh, H. L.: Med. Rec., New York, 1911, lxxx, 1309.
20. Friedenthal, H.: München. med. Wchnschr., 1911, lviii, 2411.
21. Dibbelt, W.: Berl. klin. Wchnschr., 1911, xlviii, 2058.
22. Frost, C. A.: Arch. Pediat., 1912, xxix, 55.
23. Davis, E. V.: Jour. Am. Med. Assn., 1912, lviii, 1676.

believing that the mother's toxemia may cause the death of the baby as late as three weeks after birth.

Breast milk is also of value for premature infants. Samuelson[24] concludes that the energy quotient for premature infants should be from 115 to 150. Hess[25] advises breast milk for premature infants, diluted with water or sugar water, with a caloric value of 30 the first day and increases the food 10 calories daily. If indigestion develops, the food value is kept at about 90 calories a day. At two weeks a baby weighing under 1,500 gm. should take 120 to 140 calories; one weighing over 1,500 gm. 110 to 130 calories. Artificial heat is always necessary; also one-eighth of the body weight of water should be given in twenty-four hours. Failure to gain and indigestion were noted when 140 calories per day was exceeded.

Mayerhofer and Pribram[26] report 100 infants fed successfully on breast milk preserved by the addition of hydrogen peroxid and calcium dioxid.

ARTIFICIAL FEEDING

Certified Milk.—That certified milk is the best form of cow's milk an infant can obtain is not to be denied (Porter,[27] Mosher,[28] Rice[29]). Labbe,[14] who is strongly in favor of breast feeding, notes that accidents may happen even with clean cow's milk. Delépine[30] says that, though infantile mortality is still high in Manchester (England), the reduction of the amount of tuberculous milk supplied to the city has had a distinctly beneficial effect on infant mortality, which has been markedly reduced in the last twenty years.

Methods.—While most writers (Darling[31]) still advocate percentage feeding, checked by calculation of the caloric value of the day's feedings, Abt[32] and Chapman[33] prefer simple milk dilutions. Abt[32] gets satisfactory results on milk diluted with water or a cereal, with 1 to 5 per cent. of sugar added. Elaborate and complicated formulas are not required. The careful preparation and proper administration of the food are not the least important elements in successful feeding. Chapman[33] prefers diluted whole milk. While the advantage of percentage feeding is accuracy, the main disadvantage is its difficulty of application. Levy[34]

24. Samuelson, S.: Ztschr. f. Kinderh., 1911, ii, 18.
25. Hess, J. H.: Am. Jour. Dis. Child., 1911, ii, 302.
26. Mayerhofer, E., and Pribram, E.: Ztschr. f. Kinderh., 1912, iii, 525.
27. Porter, Langley: Cal. State Jour. Med., 1911, ix, 415, and 460.
28. Mosher, G. C.: Dietet. and Hyg. Gaz., 1912, xxviii, 137.
29. Rice, C. H.: New Orleans Med. and Surg. Jour., 1911, lxiv, 370.
30. Delépine, Sheridan: Lancet, London, 1912, i, 1424.
31. Darling, E. A.: Boston Med. and Surg. Jour., 1911, clxv, 747.
32. Abt, I. A.: Chicago Med. Rec., 1912, xxxiv, 115.
33. Chapman, W. D.: Illinois Med. Jour., 1912, xxi, 580.
34. Levy, David: Jour. Am. Med. Assn., 1912, lviii, 1925.

gives one-half milk up to 6 months, two-thirds milk to 9 months and then undiluted milk. He allows five feedings at four hour intervals and limits the daily quantity to one liter of mixture. After 6 months he also allows broths, fruits and vegetables. Porter[27] also considers 32 ounces of milk the greatest daily ration any baby should have, with 8 ounces as the maximum feeding. He begins cereals and fruits at about 10 months. He starts on seven feedings a day and reduces rapidly to six or five. Grulee[35] never gives over a quart of milk in twenty-four hours and rarely over 1½ pints; that is 1½ ounces of whole milk to the pound of body weight, with ¾ to 1¼ ounces of carbohydrate, for the normal baby. The rest of the food should be cereals and vegetables. Darling[31] considers cow's milk unmodified or modified by dilution or removal of part of its fat content, a suitable infant food. Hanbidge[36] has had thirty-five infants do well on undiluted cow's milk, while only four did not do well on it. He believes that the lack of dilution greatly helps digestion. Shaw[37] states that some babies thrive on mixtures which in others would produce serious if not fatal results. The success of any system of feeding depends on the manner in which it is applied rather than on the method itself. Wieland[7] also believes that the proper administration of the food (at regular intervals and in correct amount) is as important as is the choice of the special mixture. Johnston[38] advises judging the correctness of the feeding by the weight, the temperature and the stools. Courtney[39] says one should be guided by general rules of infant feeding, paying only secondary attention to the appearance or disappearance of curds in the stools. Abt[32] believes, in order to feed an infant successfully, one must have a definite knowledge of his digestive functions and his constitutional state. Knowledge of the physiology of food, the injuries produced by food and the tolerance for food is also requisite. Overfeeding, which Porter[27] considers more common than underfeeding, is as disastrous as underfeeding. Every baby should be studied as an individual. He may be difficult to feed because he is suffering from an inherited disease, an anatomic malformation, a constitutional vice or an infectious disease. Fresh air, intelligent care, good housing, frequent bathing, cleanliness and proper clothing are powerful adjuvants in all that pertains to normal digestion and the upbuilding processes of infants. Spriggs[40] also lays stress on correct hygiene in the feeding in difficult cases. Benson[41] fed 400 infants in the Babies' Dairies of New York City on seven ascending

35. Grulee, C. G.: Journal-Lancet, 1912, xxxii, 141.
36. Hanbidge, W. B.: New York State Jour. Med., 1912, xii, 188.
37. Shaw, H. L. K.: Albany Med. Ann., 1912, xxxiii, 13.
38. Johnston, C. H.: Physician and Surg., 1911, xxxiii, 353.
39. Courtney, A. M.: Am. Jour. Dis. Child., 1912, iii, 1.
40. Spriggs, A. I.: Clin. Jour., 1911, xxxix, 57.
41. Benson, R. A.: Am. Jour. Obst., 1911, lxiv, 1084.

dilutions of the upper 16 ounces from a quart (7 per cent. fat); then on three dilutions of whole milk. Many babies can digest whole milk at 3 or 4 months of age and most infants at 6 months. He used no lime water; water as diluent for the first month, then barley water. The milk stations of the Boston Milk and Baby Hygiene Association[16] supply whole milk and various formulas. They hold weekly conferences on keeping babies well, and give talks on prophylaxis and hygiene, educational work which is giving excellent results. Davis[15] noted that bottle-fed babies from one to three months of age gave the highest mortality.

Fat.—While most physicians still keep the fat percentage cautiously low, Ladd[42] has had excellent results, even in atrophic infants, on higher percentages of fat than are ordinarily given. Stolte[43] believes that infants will thrive on any food which causes soap production in the feces, with the absence of fermentation. The essential point is the proper balance between the fat and lime salts in the intestines to bring about saponification.

Sugar.—Benson[41] reports good results on milk-sugar in his 400 cases. Ladd[42] found that, in atrophic infants, the average rate of gain on whey mixtures with lactose was 26 per cent. less than on similar whey mixtures with maltose. Schlutz,[44] after prolonged studies, concludes that lactose possesses no distinct pyrogenic action. Leopold[45] reports seven infants in whom excellent results were obtained on milk mixtures containing dextrimaltose. Neff[46] thinks that maltose will answer all requirements for infant feeding.

Mixed Carbohydrates.—Keller's malt-soup and other mixtures containing flour, oatmeal water and dextrimaltose are considered excellent by Levy,[34] Grulee,[35] Neff[46, 47] and Brady.[48] Brady has used Keller's malt-soup with success even in marasmus. After two or three months he changes to a milk mixture with high sugar. Grulee[35] describes the preparation of wheat flour and considers that fermentation is less active when this is used in place of barley flour. The various carbohydrates are absorbed one after the other. This may prevent a sudden taxing of the organism by a rapid absorption of one digestion product. Stolte[43] says "the more complex the carbohydrate, the longer is fermentation postponed." This explains the great value of mixtures of dextrimaltose and flour.

42. Ladd, Maynard: Arch. Pediat., 1912, xxix, 324.
43. Stolte, K.: Jahrb. f. Kinderh., 1911,lxxiv, 367.
44. Schlutz, F. W.: Am. Jour. Dis. Child., 1912, iii, 95.
45. Leopold, J. S.: Arch. Pediat., 1911, xxviii, 844.
46. Neff, F. C.: Medical Herald, 1912, xxxi, 24.
47. Neff, F. C.: Jour. Am. Med. Assn., 1911, lvii, 2068.
48. Brady, J. M.: Jour. Am. Med. Assn., 1912, lviii, 751.

Proteid.—Hunt[1] details the methods employed to aid protein digestion; boiling the milk, adding cereals, adding alkalies in small quantity, partially peptonizing, using whey, buttermilk or equal parts of human and cow's milk. Each child must be considered as an individual and the method adopted must be the one best fitted to the needs of the particular infant. Porter[27] gives 1¼ ounces of protein-containing fluid per pound per day. Boggs[49] advises split protein for babies under 7 months; then he changes to top milk mixtures. Ruhräh[50] reports success from soy bean in the form of a dilute gruel, with or without milk added. It may be of use when milk disagrees. Each ounce of soy bean flour yields about 13 gm. of protein and 120 calories.

Albumin Milk.—The Germans report excellent results on Finkelstein's albumin milk. Benfey[51] gave it to forty-four normal infants from birth, all doing well, and to thirty-nine new-born infants with infections, 71 per cent. of whom did well. He gives 150 to 200 gm. for each kilogram of body weight daily, with malt-sugar up to 6, 7 or 8 per cent. Beck[52] gave it to 175 infants, with only twelve deaths. The quantity of food and the sugar in it should be rapidly increased. Haimis[53] found it of value in chronic nutritional disturbances. Schlutz[54] has seen only good results on it in diarrhea; Heiman[55] observed some improvement on it, while Neff[47] considers that it has a limited field in cases of catarrhal enteritis. Morse[56] reports eight infants fed on a laboratory milk with high casein and malt-sugar. He considers that withdrawing the lactose and increasing the percentage of casein, followed by adding dextrimaltose, is a valuable method of treating intestinal disturbances associated with fermentation in infancy. As babies will not thrive on such mixtures, they should be discontinued as soon as symptoms are relieved. He believes that this method is not generally applicable. Heim and John[57] fed a milk made by adding the fat and casein from ⅔ liter of raw milk mixed with ⅔ liter of water, to ⅓ liter of boiled milk, to five infants, all of whom gained in weight and did well.

Lactalbumin.—Sill[58] added powdered lactalbumin to milk mixtures for infants who were ill-nourished and markedly under weight, all of them gaining well on it. Bornstein[59] performed numerous metabolism

49. Boggs, W. D.: Calif. State Jour. Med., 1911, ix, 496.
50. Ruhräh, John: Arch. Pediat., 1911, xxviii, 841.
51. Benfey, Arnold: Jahrb. f. Kinderh., 1912, lxxv, 280.
52. Beck, Carl; Jahrb. f. Kinderh., 1912, lxxv, 315.
53. Haimis, G.: Gyógyászcat, 1911, No. 15.
54. Schlutz, F. W.: Journal-Lancet, 1912, xxxii, 181.
55. Heiman, H.: Arch. Pediat., 1911, xxviii, 964.
56. Morse, J. L.: Am. Jour. Dis. Child., 1911, ii, 315.
57. Heim, P., and John, K. M.: Ztschr. f. Kinderh., 1912, iv, 1.
58. Sill, E. M.: New York Med. Jour., 1912, xcv, 1093.
59. Bornstein, K.: Arch. f. Kinderh., 1911, lvi, 16.

experiments on three infants using a proprietary form of lactalbumin. He concludes that it causes more frequent and less consistent stools, but that, in spite of the increased quantity of protein ingested, the excretion of feces, as well as of calcium and nitrogen, is not changed.

Boiled Milk.—Ladd[42] notes that babies on boiled milk did as well as those on the same mixtures unboiled. Abt[32] thinks that scalded or boiled milk sometimes agrees when raw cow's milk causes indigestion and diarrhea. Hunt[1] finds that artificial gastric digestion experiments show that boiling the milk decreases the amount of soluble calcium salts and removes any carbon dioxid present. Rudolf,[60] who advocates low percentages in infant feeding, favors using boiled milk and quotes many English authorities who have used undiluted sterilized cow's milk successfully. Verley[61] reports a ten-day-old infant who, failing to gain on milk and barley-water, did well on whole milk, sterilized. Benson,[41] in his series of 400 babies, neither pasteurized nor sterilized any of his formulas. Schorer and Rosenau[62] believe that all commercial pasteurizing plants should be under official control and standardized. The best temperature to meet practical conditions is 145 F. for thirty to forty-five minutes. While Schorer[63] considers it safest to pasteurize in the sealed bottle, Schulz[64] found that as much as 0.03 gm. of silicic oxid passed into the milk when it was sterilized in the sealed bottle. He thinks this may help to explain the obstinate constipation and possibly infantile scurvy which may occur. Jordan[65] notes an instance of infantile scurvy occurring in a baby fed only on sterilized milk.

Desiccated Milk.—Nageotte-Wilbouchevitch[66] and Aviragnet, Bloch-Michel and Dorlencourt[67] have had success with dried milk in powdered and tablet form. The former used it for children who could not take milk without symptoms of intoxication. The latter have employed it for three years in both well and ill infants. It was most successful in cases of enteritis with vomiting. The powder contains less than 4 per cent. water. There are three varieties of desiccated milk, made from whole and skimmed milk. But prolonged feeding of such dried, sterile food is exceedingly dangerous.

Buttermilk.—Neff[68] advises buttermilk in marasmus, chronic colitis, the "exudative diathesis" and difficult feeding cases. It is, however, not

60. Rudolf, R. D.: Canadian Med. Assn. Jour., 1912, ii, 173.
61. Verley, R. G.: British Med. Jour., 1912, i, 831.
62. Schorer, E. H., and Rosenau, M. J.: Jour. Med. Research, 1912, xxvi, 127.
63. Schorer, E. H.: Am. Jour. Dis. Child., 1912, iii, 226.
64. Schulz, H.: München. med. Wchnschr., 1912, lix, 366.
65. Jordan, W. R.: Brit. Jour. Child. Dis., 1911, viii, 520.
66. Nageotte-Wilbouchevitch: Bull. Soc. de pédiat. de Paris, 1912, xiv, 23.
67. Aviragnet, E. C., Bloch-Michael, L., and Dorlencourt, H.: Bull. Soc. de pédiat. de Paris, 1912, xiv, 109.
68. Neff, F. C.: Jour. Missouri State Med. Assn., 1911, viii, 160.

a substitute for sweet milk. It is well tolerated in hot weather. Clean, fresh milk must be used for preparing the buttermilk and top milk may be added gradually, if desired. When breast milk is not available, Neff[47] uses buttermilk in cases of fat intolerance and enterocolitis. Heinemann[69] states that many of the ferments sold for making sour milk are worthless. The fermented milks are only of value in infancy because of their low fat content.

Vacuum Bottles.—Tonney and Pillinger[70] advocate the use of the vacuum bottle in infant feeding, if controlled by a thermometer. It not only keeps the milk warm, but effectually pasteurizes it. It will maintain a proper temperature (about 115 F.) for ten hours. If the milk, on testing with the thermometer, is found under 115 F., it should not be used.

Card for Calculating Mixtures.—Young[71] has devised a card which reduces mathematics to a minimum. The celluloid envelope contains two celluloid cards, printed on each side, for making 20, 32, 40 and 48 ounce mixtures, with milk and cream, or with whey. Any formula can rapidly be worked out with this card, together with the total caloric value of the mixture. The card is to be commended because no attempt is made to suggest any formula for a baby of any age or weight.

Caloric Values.—Fraley's[72] formula for calculating the total caloric value of percentage mixtures is exceedingly simple. He adds together twice the fat, the sugar and the protein; and then multiplies by 1¼ times the total quantity ingested in twenty-four hours, with a result which is the total caloric value of the day's feedings. $(2F+S+P)\times 1\frac{1}{4}Q=C$. Farr[73] has collected all the foods used in childhood and demonstrates their comparative caloric values exceedingly well, both by photographs and tables. English[74] has prepared detailed diet lists for children from 2 to 15 years of age, advising regular meals, sufficient mastication, little fluid at meals and nothing between meals except water. Stargardter[75] has made several feeding and metabolism experiments to show the amount of food and of protein required by children. He gives tables showing the number of calories necessary and diets for children of two and seven years. Hoobler[76] gives a list of foodstuffs containing phosphorus, potassium, sodium, iron, sulphur, chlorin, magnesium and calcium, noting the percentage of the mineral salt in each kind of food. He suggests using these foodstuffs therapeutically in diseases in which there is a

69. Heinemann, P. G.: Jour. Am. Med. Assn., 1912, lviii, 1252.
70. Tonney, F. O., and Pillinger, H. H.: Jour. Am. Med. Assn., 1912, lviii, 1495.
71. Young, J. H.: Boston Med. and Surg. Jour., 1912, clxvi, 372.
72. Fraley, F.: Arch. Pediat., 1912, xxix, 123.
73. Farr, Clifford B.: Arch. Pediat., 1912, xxix, 110.
74. English, D. E.: Jour. of the Med. Soc. of New Jersey, 1911, viii, 354.
75. Stargardter, Julius: Arch. f. Kinderh., 1912, lvii, 305.
76. Hoobler, B. R.: Arch. Pediat., 1912, xxix, 208.

known deficiency in mineral salts. Those containing salts of iron will be employed with least difficulty, since they are of use in secondary anemia.

DISORDERS OF THE DIGESTIVE SYSTEM

Stomatitis.—Kerr[77] divides stomatitis into acute catarrhal stomatitis, stomatitis mucosa, stomatitis unaccompanied by odor or ulceration, stomatitis with ulceration and no offensive odor, syphilitic stomatitis, stomatitis ulcerosa, stomatitis gangrenosa and stomatitis with membrane formation. Gaujoux[78] differentiates between simple aphthous stomatitis and "foot and mouth disease." In the former, due to some error in diet, the treatment is a mild antiseptic wash. In the latter, which is generally traceable to an infected milk-supply, all milk should be boiled, and sodium salicylate and occasionally diphtheria antitoxin should be administered.

Foreign Bodies in Esophagus.—Abrand[79] reports the cases of two children who swallowed pieces of bone about an inch long, which lodged in the esophagus. The first case was a boy of 12 years, who vomited the bone two days later. In the second case the bone was readily removed under anesthesia.

Pyloric Stenosis.—Leavitt and Porter[80] studied twenty-four infants with muscular pyloric hypertrophy. They conclude that those babies who retain the regurgitant type of vomiting do better than those in whom the vomiting is constantly cumulative and propulsile. They were impressed with the need of a uniform method of studying and reporting cases of congenital pyloric spasm and hypertrophy. Lapage[81] outlines the medical treatment as careful regulation of feeding, with easily digested food, in small quantities at first; lavage; saline infusion by rectum or subcutaneously; brandy; cod-liver oil inunctions and perhaps opium or belladonna. Bilderback[82] reviews the subject. Operative cases are reported by Gavin,[83] a baby of 7 weeks recovering after posterior gastrojejunostomy; Bunts,[84] seven cases, with four recoveries; Cotiguola,[85] three cases with one recovery; Katzmann and Abell,[86] an infant of 6 weeks who died thirty-one hours after pyloroplasty; Smedley,[87] an infant of 6 weeks, with death from enterocolitis two weeks after posterior gastro-

77. Kerr, Legrand: Am. Med., 1912, xviii, 108.
78. Gaujoux, E.: Ann. de méd. et de chir. infant., 1912, xvi, 1.
79. Abrand, H.: Bull. Soc. de pédiat. de Paris, 1912, xiv, 16.
80. Leavitt, W. B., and Porter, L.: Jour. Am. Med. Assn., 1912, lviii, 256.
81. Lapage, C. P.: Practitioner, London, 1912, lxxxviii, 401.
82. Bilderback, J. B.: Medical Sentinel, 1911, xix, 744.
83. Gavin, G. E.: Southern Med. Jour., 1912, v, 241.
84. Bunts, F. E.: Am. Jour. Med. Sc., 1912, cxliii, 14.
85. Cotiguola, M. F.: Semana méd., 1912, xix, 489.
86. Katzmann, E. F., and Abell, T.: Kentucky Med. Jour., 1911, ix, 804.
87. Smedley, R. C.: Northwest Med., 1912, iv, 5.

enterostomy. Tanaka[88] reports two more fatal cases. Hoffa[89] reports five interesting cases of visible gastric peristalsis, without vomiting or pyloric stenosis. He concludes that visible gastric peristalsis does accompany pylorospasm and does not necessarily denote pyloric stenosis. Graham[90] reports a case of persistent vomiting in a boy of 6½ years in whom no lesion of the pylorus or stomach was found post mortem. Sarcoma of one eye had been operated on two months before vomiting began and sarcoma of the liver was found at autopsy. Graham believes that pyloric stenosis occurs in children and young adults more commonly than is supposed; that it may remain latent for years; that the entire disappearance of all symptoms and the apparent health of the infant during childhood suggest the probability of absorption of the hypertrophy.

Habitual Vomiting.—Lowenburg[91] considers irregular feeding and overfeeding responsible for the vast majority of cases of functional vomiting in infants under 1 year of age. In older children some indiscretion in diet is the common cause of vomiting. Cheinisse[92] considers habitual vomiting in infants generally due to overfeeding; but it may be due to insufficient nourishment or to nervous hyperexcitability of the gastric mucosa, with or without pyloric stenosis. Hahn[93] found that less dilute food, such as milk, barley-jelly and sugar, containing 1,000 calories to the liter, was well borne by those infants in whom no organic cause for the vomiting could be found. Kevacs[94] also advises more consistent foods (milk or strained vegetables) rather than a diluted milk mixture. Smith[95] describes seven cases of nervous vomiting, occurring in neurotic children with enuresis, migraine, habit-spasm or rheumatism. The nervous inheritance is always present, associated with some other functional condition. Recovery follows good hygiene, elimination of the cause aggravating the underlying nervous instability and firm moral control.

Recurrent Vomiting.—Sedgwick,[96] as a result of his investigations, found that the creatin metabolism, the endogenous nitrogen metabolism, is abnormal during attacks of recurrent vomiting. In many cases the removal of adenoids has been followed by cessation of the attacks of recurrent vomiting. Morris[97] calls attention to "prune juice" vomiting as a symptom of the cases of recurrent vomiting which he has observed.

88. Tanaka, Tamio: Jahrb. f. Kinderh., 1912, lxxv, 18.
89. Hoffa, T.: Monatschr. f. Kinderh., 1912, x, 523.
90. Graham, E. E.: Am. Jour. Dis. Child., 1911, ii, 407.
91. Lowenburg, H.: Jour. Am. Med. Assn., 1912, lviii, 180.
92. Cheinisse, L.: Semaine méd., 1911, xxxi, 565.
93. Hahn, H.: Med. Klin., 1911, No. 38.
94. Kevacs, B.: Gyógyászcat, 1911, No. 43.
95. Smith, Eric B.: Lancet, London, 1911, ii, 1769.
96. Sedgwick, J. P.: Am. Jour. Dis. Child., 1912, iii, 209.
97. Morris, R. S.: Am. Jour. Obst., lxv, 534.

Nesbitt[98] reports the case of a baby thirty hours old who vomited bright red blood; then passed dark masses in the stools up to 3 days of age; who grew up well afterward. Hess[99] advises the duodenal catheter (a soft rubber Nélaton catheter, No. 15) in the differential diagnosis between cases of vomiting due to pylorospasm and those cases not due to spasm of the pylorus. Cardiospasm often accompanies pylorospasm. Duodenal feeding is of value in cases of persistent vomiting with marked inanition. Lust[100] reports a case of "rumination" in an infant of 5½ months, with increasing malnutrition and death at 8 months.

Liver: Jaundice.—Hess[101] observed 124 infants with icterus neonatorum. By using the duodenal catheter he found that bile was excreted during the first twelve hours of life. During the next twenty-four hours the amount excreted is profuse if icterus is present; scanty or absent when there is no jaundice. The function of bile excretion becomes fully established during the first week or ten days. The appearance of jaundice precedes the excretion of bile into the duodenum. Raynaud and Coudray[102] report a case of catarrhal jaundice in a girl of 4 years, with a typical tetragenus found in blood cultures, which was agglutinated by the patient's blood-serum. Talbot[103] reports a case of acute intestinal indigestion in a boy of 10 years, ill six weeks, eating ten or more apples daily. For two weeks fever and headache had been noted, while jaundice appeared one week previously. On excluding fat and sugar from his diet, he gained 5 pounds in twelve days, his food being restricted to meat, starches and skimmed milk. Urobilinogen was absent from the urine while there was complete obstruction to bile, which then appeared in excessive amounts when the obstruction was removed.

Cirrhosis.—Lahiri[104] reports a case of cirrhosis of the liver in an infant 18 months old, ill eight months. There was marked ascites. On three injections into the cellular tissue of the flank of the child's own serum, first ½ c.c.; eight days later 1 c.c., and five weeks later 2 c.c.; complete recovery followed. Pagliano and de Luna[105] report the case of a boy of 12 years, with hepatic cirrhosis, without any history of syphilis, malaria or alcohol. The condition had persisted for years, without the boy having grown up. Sukares[106] reports the autopsy on a child of 2 years in whom the diagnosis of tuberculosis had been made. The only lesion found was hepatic cirrhosis of the rare microsplenic, anicteric type.

98. Nesbitt, M. D.: Australasian Med. Gaz., 1911, xxx, 514.
99. Hess, A. F.: Am. Jour. Dis. Child., 1912, iii, 133.
100. Lust, F.: Monatschr. f. Kinderh., 1911, x, 316.
101. Hess, A. F.: Am. Jour. Dis. Child., 1912, iii, 304.
102. Raynaud, M., and Coudray, M.: Progrès méd.., 1911, 206.
103. Talbot, F. B.: Am. Jour. Dis. Child., 1912, iii, 398.
104. Lahiri, M.: Practitioner, London, 1912, lxxxviii, 478.
105. Pagliano and de Luna: Ann. de méd. et chir. Inf., 1912, xvi, 212.
106. Sukares, O.: Gyógyászcat, 1911, No. 44.

Acute Yellow Atrophy.—Two fatal cases are reported, one by Phillips[107] in a girl of 5 years, who died in one week; the other by Drennan[108] in a boy of 8 years, with death in one month.

Appendix.—McPherson[109] describes an appendix found at the autopsy of a 15-hour-old baby. It was adherent by many fibrous bands to the posterior aspect of the cecum and assumed a horse-shoe shape. Kee[110] has removed the appendix from twenty-three children between 2½ and 13 years of age. In twenty cases the appendix was perforated, gangrenous or ulcerated, with abscess; six of these died. Canaguier and Hamel[111] report the removal of the appendix from an infant five days old, with death a week later. Rheindorf[112] found oxyuris in the appendix in six out of thirteen bodies of children between 3 and 12 years of age who died from acute appendicitis. He thinks more efforts should be made to keep the intestines of children free from seat-worms.

Intussusception, Etc.—Kakels[113] reports operating on the third day on a girl of 5 years with intussusception, who was well in three weeks. He resected 12 inches of gangrenous intestine, the ileum having passed through the ileocecal valve. An adenoma was found in the intestine at the apex of the intussusception. King[114] reduced an intussusception in a boy of 11 weeks by operating eight hours after the onset of the attack, also removing a strangulated appendix. Recovery was uneventful. Dunn[115] reports operating after six days of illness on a boy of two years, resecting 6 inches of small intestine, with death in forty-eight hours. A Meckel's diverticulum, 2 inches long, had invaginated into the small intestine. Guinon and Fauquez[116] report late operation, with death, on an infant of 5½ months with acute invagination. Weill-Hallé[117] reports another fatal case in an infant of 11 months, ill only twenty-four hours; Grisel,[118] one case in an infant of 7 months who died two days after operation and another, a boy of 12 years, who recovered without operation. O'Neill[119] reports the history of a colored boy of 15 months, in whom what was supposed to be intussusception proved to be a small china pig the child had swallowed. This was passed successfully, complete recovery

107. Phillips, John: Am. Jour. Med. Sc., 1912, cxliii, 177.
108. Drennan, A. M.: Jour. Path. and Bacteriol., 1911, xvi, 141.
109. McPherson, Ross: Bull. of the Lying-In Hosp., City of New York, 1912, viii, 67.
110. Kee, A. L.: West. Med. Rev., 1912, xvii, 125.
111. Canaguier and Hamel: Bull. Soc. de pédiat., Paris, 1912, xiv, 45.
112. Rheindorf: Berl. klin. Wchnschr., 1912, xlix, 450 and 503.
113. Kakels, M. S.: Am. Jour. Surg., 1911, xxv, 363.
114. King, J. C.: Southern Cal. Practitioner, 1912, xxvii, 57.
115. Dunn, L. A.: Clin. Jour., 1912, xxxix, 350.
116. Guinon and Fauquez, Bull. Soc. de pédiat. de Paris, 1911, xiii, 361.
117. Weill-Hallé: Bull. Soc. de pédiat. de Paris, 1911, xiii, 369.
118. Grisel, P.: Bull Soc. de pédiat. de Paris, 1911, xiii, 436.
119. O'Neil, Owen: Yale Med. Jour., 1911, xviii, 254.

resulting. He also reports two cases of intussusception with operation, in a girl of 4 months who died of enterocolitis fourteen days later, and a boy of 8 months who recovered. Comby[120] reports a case of chronic invagination in a girl of 4 years, in whom the condition was supposed to be ileocolitis. She was ill twenty-six days, suffering from pain, vomiting and diarrhea, with blood and mucus in the stools. Triboulet and Savariaud[121] describe another case of chronic invagination in a boy of 5½ years, easily reduced at operation. Matheny[122] reports eight cases of intussusception with only two deaths after operation. Early diagnosis and operation are essential to a low mortality. As the mesentery in childhood is long, violent pitching or swinging of children should be avoided. He also lays stress on digital examination per rectum whenever a child has abdominal pain. Other causes of abdominal pain in children are given by Finkelstein[123] as appendicitis, disease of the large or small intestine, neuralgia of the spinal nerves due to caries, dyspepsia, intestinal stenosis following circumscribed tubercular peritonitis, and renal colic. Kirmisson[124] describes excising a persistent Meckel's diverticulum which opened at the umbilicus, in an infant of 1 month, with recovery. Waugh[125] reports a rare case of congenital hernia of the cord, causing acute intestinal obstruction. Spinal anesthesia was used and enterectomy (lateral anastomosis) done. While the baby recovered from the operation, it died a month later of marasmus. Wilcox[126] operated on an infant 48 hours old, who vomited continually. The entire large intestine was found to be only a fibromuscular cord. Death followed two hours after operation. Hauser[127] found total atresia of the duodenum at autopsy in a baby 7 days old who had vomited persistently since birth. Kermanner[128] also describes a case of congenital atresia of the duodenum. A case of fatal duodenal ulcer in an infant of one month, who died of intestinal hemorrhage, is described by Weill and Gardère.[129] Sheffield[130] describes a monstrosity in which no anus could be found. Law[131] reports two cases of imperforate anus in girls of 3½ and 13 years, the intestine opening into the vagina. While the larger one recovered after operation, the

120. Comby, Jules: Arch de méd. des enf., 1912, xv, 47.
121. Triboulet and Savariaud: Bull. Soc. de pédiat. de Paris, 1912, xiv, 136.
122. Matheny, A. R.: Pennsylvania Med. Jour., 1912, xv, 440.
123. Finkelstein, F.: Zentralbl. f. Kinderh., 1912, xvii, 1.
124. Kirmisson, E.: Bull. de l'académie Méd., 1912, lxviii, 143.
125. Waugh, George E.: Lancet, London, 1912, I, 427.
126. Wilcox, G. A.: Jour. Med. Assn. of Georgia, 1911, i, 145.
127. Hauser, Hans: Monatschr. f. Geburtsh. und Gynäk., 1911, xxxiv, 678.
128. Kermanner, F.: Virchows Arch. f. path. Anat., 1912, ccvii, March.
129. Weill and Gardère: Ann. de méd. et chir. inf., April 15, 1912, xvi.
130. Sheffield, H. B.: Med. Rec., New York, 1911, lxxx, 1164.
131. Law, A. A.: Journal-Lancet, 1912, xxxii, 202.

smaller died in thirty-five days from septic nephritis. Farnsworth[132] reports two similar cases, in girls of 3 weeks and 3 years.

Megacolon.—Three cases are described in recent German literature, all successfully treated. Göppert's[133] case was a boy of 3½ years with constipation and enormous distention of the colon, due to a bend in the descending colon which prevented defecation. The daily passage of a rectal tube during several months was followed by regular normal defecation since. Hoffmann[134] describes two cases in which resection of the sigmoid and upper rectum was performed, with recovery. In one, a boy of 14, 30 cm. of intestine was resected. Sheffield[130] also operated on his patient, with death the next day. He found the descending colon widely dilated, cornet shaped, with its wide extremity (6½ inches in circumference) terminating at the sigmoid.

Enteroptosis.—Smith[135] studied 109 girls under 13 years of age and found that children with a tendency to enteroptosis show frailness, lack of fat, slenderness of muscle and lack of vigor in body development. He recommends special attention to frail children very early, with continual treatment to make them more robust. Marfan[136] describes a condition which he calls the "large flaccid abdomen of infants," found in babies with rickets, but to be distinguished from the large tympanitic abdomen of infancy. It is an atonic condition of the abdominal muscles and is due to recurrent attacks of catarrhal gastro-enteritis. At autopsy the intestine is found to be from ⅓ to ½ as long as is normal, from atony of the intestinal musculature, analogous to the gastric dilatation also present in most of these infants.

Constipation.—Périer and Gaujoux[137] consider constipation due to obstruction, to modification of the contents of the intestine and to defective action of the muscles or nerves of the intestines. They found that congenital stenosis of the rectum may escape discovery for weeks or even months. Ward[138] reports twelve children with joint tuberculosis, one with rheumatoid arthritis and one with ulcerative colitis, all with marked intestinal stasis. He performed ileocolostomy on all with recovery.

Diarrhea.—Forsyth[139] considers infantile diarrhea due to an infection following overfeeding or irregular feeding. Bilderback[140] states that impure milk causes diarrhea; heat and overfeeding or improper feeding

132. Farnsworth, C. P.: Med. Rec., New York, 1912, lxxxi, 470.
133. Göppert, F.: Berl. klin. Wchnschr., 1912, xl, 588.
134. Hoffmann, H.: Beitr. z. klin. Chir., 1911, lxxvi, 533.
135. Smith, R. R.: Jour. Am. Med. Assn., 1912, lviii, 385.
136. Marfan, A. B.: Arch. de méd. des enf., 1911, No. 8.
137. Périer, E., and Gaujoux: Ann. de méd. et chir. inf., 1911, xv, 593.
138. Ward, E. B.: Practitioner, London, 1912, lxxxviii, 570.
139. Forsyth, R. L.: Australian Med. Jour., 1912, ii, 289.
140. Bilderback, J. B.: Northwest Med., 1911, iii, 310.

aggravate it. Salle[141] has performed experiments on animals to show the direct injury done to the gastric function by heat. As even slight changes in the food may be dangerous when the function of the digestive apparatus is disturbed by hot weather, less food should be given and more water is necessary. Wood[142] considers the cause of summer diarrhea to be the high bacterial content of the milk. Grulee[35] considers milk-sugar responsible for many cases of summer diarrhea.

Rolleston and Molony[143] describe eleven cases of fatal purpura complicating infectious diarrhea. Symptomatic purpura occurs mainly on the abdomen and chest in infants under 1 year of age. It is usually a terminal phenomenon in prolonged cases. The prognosis is extremely grave.

Synnott[144] lays stress on pasteurizing or sterilizing even a clean milk in hot weather. Dennett[145] advises a milk and water mixture, boiled, without any sugar, for poorly nourished infants. The well nourished babies do best on barley gruel for a few days. Stephens[146] gives water only, then cereals or albumins and finally milk. Synnott[144] advises water in abundance, intestinal irrigation in many cases, isolation of the baby and disinfection of all sick-room implements. Machell[147] makes a plea for the short rectal tube, since a larger amount can be injected and it is retained longer when a short nozzle is used. Albumin milk is advised by Stolte,[43] Neff,[47] Beck,[52] Schlutz,[54] Heiman[55] and Dennett.[145] Buttermilk is also advised by Neff[47] and Stolte.[43] Brady[48] recommends malt-soup in diarrhea. Ruhräh[50] recommends soy bean flour. Nageotte-Wilbouchevitch[66] and Aviragnet, Bloch-Michel and Dorlencourt[67] had good results with desiccated milk in powder and tablet form.

Synnott[144] gives castor oil and advises antidysenteric serum in certain cases. Dennett[145] believes that cathartics should always be given to babies with diarrhea with great discretion. He recommends them in the infectious diarrheas, with starvation for forty-eight hours only. Wood[142] uses no drugs except opium when necessary. Hand[148] speaks of the value of bismuth salicylate as an intestinal antiseptic in acute diarrhea. Thymol and salol are too irritating and calomel is not advised. Grulee and Buhlig[149] conclude from their observations on milk that sodium benzoate (1 grain to 6 ounces of milk) must have a slight effect in inhibiting some kinds of bacteria, because of the difference in the time the milks

141. Salle, V.: Jahrb. f. Kinderh., 1911, lxxiv, 697.
142. Wood, A. J.: Australian Med. Jour., 1912, ii, 263 and 278.
143. Rolleston, H. D., and Molony, J. B.: Brit. Jour. Child. Dis., 1912, ix, 1.
144. Synnott, M. J.: Arch. Pediat., 1911, xxviii, 989.
145. Bennett, Roger H.: Med. Rec., New York, 1911, lxxx, 1138.
146. Stephens, H. D.: Australian Med. Jour., 1912, ii, 288.
147. Machell, H. T.: Arch. Pediat., 1911, xxviii, 837.
148. Hand., Alfred Jr.: Arch. Pediat., 1912, xxix, 148.
149. Grulee, C. G., and Buhlig, W. N.: Arch. Pediat., 1911, xxviii, 849.

become sour and coagulated; that sodium benzoate seems to be a fair preservative only when very few living bacteria are present. From their clinical observations on nine infants from a few weeks to almost 2 years of age, they found that 2½ to 5 grains of sodium benzoate in twenty-four hours produced no recognizable symptoms, even though the children were suffering from gastro-intestinal disturbances of a serious nature. Ellis[150] reports good results in the chronic forms of diarrhea on magnesium sulphate. Löbisch[151] treated seven infants with summer diarrhea, aged 4 to 13 months, with hypertonic saline solution given intravenously to six and subcutaneously to one, with recovery. Rogers[152] also recommends hypertonic saline injections, given intravenously, controlled by estimation of the specific gravity of the blood, in infantile diarrhea.

Infantile Atrophy.—Bar[153] considers athrepsia due to lack of production of ferments in the infant stomach. Cow's milk is an alien substance and acts as a poison unless ferments are present to modify it. The child becomes sensitized and the phenomena of anaphylaxis result. Sterilizing the milk will not prevent this action. Rarely, after prolonged illness during pregnancy, a mother's milk may also poison her baby. Czerny[154] considers atrophy not a disease *per se* but a disturbance in growth and general nutrition which is the result of nutritional disturbances or of infectious processes or of both. When the condition is a nutritional disturbance alone, splendid results may be obtained by dietetic management. The occurrence of atrophy is distinctly the result of a constitutional anomaly. Three classes of children suffer from it — those who inherit the "exudative diathesis," those of neuropathic or psychopathic parents and those with the "hydropic constitution." Atrophy is characterized by a cessation of normal growth throughout the entire organism, followed by a failure of the development of fat. Chapman[33] considers nutritional disturbances in infants either as disorders of alimentation, due to improper food, indigestion and failure of assimilation; or as disorders of infection, due to lack of cleanliness. Variot and Morancé[8] report twelve cases to show that the abdominal ectasia found in atrophic infants is due to hypo-alimentation. Both large and small intestines are distended. These infants need more food than heavier children and should be fed according to age and not to weight. Ladd[42] finds that the energy quotient is greatest when the weight development

150. Ellis, H. A.: Australasian Med. Gaz., 1912, xxxi, 30.

151. Löbisch, W.: Wien. klin. Wchnschr., 1911, xxiv, 1640.

152. Rogers, Leonard: Brit. Med. Jour., 1911, ii, 1404.

153. Bar, P.: Revue Mensuelle de Gynécologie, d'Obstetrique et de Pédiatrie, 1912, vii, 161.

154. Czerny, Ad.: Am. Jour. Dis. Child., 1912, iii, 170.

is farthest from the average normal infant as determined by the weight chart. The quantity of food to be given an atrophic infant is only a little less than that which the normal infant of the same age receives and is often 1½ to 2 ounces more than would be given the normal infant of the same weight.

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